GELDRWEKIRLRPGGKKKYK

QUERY	GELDRWEKIRLRPGGKKKYK
CONSENSUS_A	-k--a----------r
A.KE.Q23-CXC-CG	-KF-A----------R
A.SE.SE6594	-K--A----------R
A.SE.SE7253	-K--A----------R
A.SE.SE7535	-K--A----------Q-R
A.SE.SE8131	-K--A----------N---R
A.SE.SE8538	-R--A----------R
A.SE.SE8891	EKK-A---M----------
A.UG.92UG037	-K--A----------R
A.UG.U455	KK--S----------N---R
CONSENSUS_B	--------------------
B.AU.AF128998	-K--K----------T-Q
B.-.NL43E9	----K----------L--
B.AU.MBC18	-K----------
B.AU.MBC200	----------Q-R
B.AU.MBC925	-------R-----R-----Q
B.AU.MBCC54	----------Q
B.AU.MBCC98	----------Q
B.AU.MBCD36	E----------R---Q
B.CN.RL42	-Q----------R
B.DE.D31	----------R
B.DE.HAN	----K----------Q
B.ES.89SP061	-G----------R
B.FR.HXB2	--------------------
B.GA.OYI	----K----------Q
B.GB.CAM1	----K----------
B.GB.MANC	-K----------
B.JP.JH31	--------------------
B.NL.3202A21	----K----------R--
B.TW.LM49	----K--RV----------R
B.US.85WCIPR54	----------
B.US.AD8	-K----------
B.US.BC	-K--K----------
B.US.DH123	-K--S----------
B.US.JRCSF	----------R
B.US.JRFL	-K--K----------R
B.US.MNCG	-------N----------
B.US.NC7	-D----------M
B.US.NY5CG	----K----------Q-R
B.US.P896	--------------------
B.US.RF	-K--K--------R---R--
B.US.SF2	----K----------
B.US.WC001	----------
B.US.WEAU160	----------N----
B.US.WR27	----K----------R
B.US.YU2	----K----------Q-R
CONSENSUS_C	-K--k----------h-m
C.BR.92BR025	-K--A--R-K-K-----H-M
C.BW.96BW01B22	-K--Q----------C-M
C.BW.96BW0402	-K--A----------Q-R
C.BW.96BW0502	EK--K----------H-M
C.BW.96BW1104	-K--T----------R-M
C.BW.96BW1210	EK--T----------R-M
C.BW.96BW15B03	EK--T-----S------C-M
C.BW.96BW1626	-K--K----------R-M
C.BW.96BW17A09	-K--T----------H-M
C.ET.ETH2220	EK--A----K-------H-M
C.IN.93IN904	EK--K----------H-M
C.IN.93IN905	-K--K----------H-M
C.IN.93IN999	EK--K--R---------H-M
C.IN.94IN11246	-K--K----------H-M
C.IN.95IN21068	-K--K----------R-M
CONSENSUS_D	-K--a----------r
D.CD.84ZR085	-K--A----------
D.CD.ELI	-K--K----------R
D.CD.NDK	-K--T--R----------A
D.CD.Z2Z6	-K--A----------R
D.UG.94UG1141	-K--E----------R
CONSENSUS_F	-K--A----------r
F.BR.BZ162	-K--A----------R
F.CD.VI174	-K--A----Q---------R
F.RW.VI69	-K--A----------R----
CONSENSUS_F1	-K--a----------r
F1.BE.VI850	-K--E----Q-------R--
F1.BR.93BR020.1	-K--A----------R
F1.FI.FIN9363	-K--A----------Q-R
F1.FR.MP411	-K--A--R----------R
CONSENSUS_F2	-K--A------?-----?-R
F2.CM.MP255	-K--A------K-----R-R
F2.CM.MP257	-K--A----------R
CONSENSUS_G	-K--A----------x---x
G.BE.DRCBL	-K--A----------R-R
G.FI.HH8793	-K--A----------R
G.NG.92NG083	-K--S----------R----
G.SE.SE6165	-K--A----------R-S--
CONSENSUS_H	-K--A----------R
H.BE.VI991	-K--A----------R---R
H.BE.VI997	-R--TL----------R
H.CF.90CF056	-K--A----------R
CONSENSUS_J	-K--D----------?-R
J.SE.SE9173	-K--D----------Q-R
J.SE.SE9280	-K--D----------R
CONSENSUS_K	-K--?----------r
K.BE.VI325	-K--T----------S---R
K.CD.EQTB11C	-K--K----Q---------R
K.CM.MP535	-K--A----------
N.CM.YBF30	-K--Q--S-Y---------R
CONSENSUS_O	SK--A--?---?--S--?-R
O.CM.ANT70C	SK--A--Q---K--S----R
O.CM.MVP5180	SK--A--R------S--A-R
CRF01-AE.CF.90CF40	-K--A----------Q-R
CRF01-AE.TH.93TH25	-K--A----------
CRF01-AE.TH.CM240	-K--A----------R---R
CRF01-AE.TH.TH022	-K--A----------R---R
CRF01-AE.TH.TH047	-K--A----------R---H
CRF02_AG.FR.DJ263	-K--S----------R
CRF02_AG.FR.DJ264	-K--S--------A-----R
CRF02_AG.NG.IBNG	-K--A----------R
CRF03_AB.RU.KAL15	-K--A----------E--R
CRF04_cpx.CY.94CY0	-K--A--R----------R
CRF04_cpx.GR.97PVC	-K--A--R----------R
CRF04_cpx.GR.97PVM	-R--A----------R-R
AC.ET.E3099G	-K--T----------N---R
AC.IN.21301	-K--K----------H-M
AC.RW.92RW009	-K--A----K-K-----T-M
AC.SE.SE9488	-K--A----------R
AC.ZM.ZAM174-21	-K--T----------S-R-M
AC.ZM.ZAM184	-K--A----------Q-R
AC.ZM.ZAM716-17	-K--A----------Q-R
ACD.SE.SE8603	-K--A----------R
AD.SE.SE6954	ER--E----Q-------R-R
AD.SE.SE7108	-K--A----------R----
ADHU.NO.NOGIL3	-K--K----------Q-R
ADU.CD.MAL	-K--A----------R
AG.NG.G3	-K--A----------R
AG.SE.SE7812	-K--A----------R
AGHU.GA.VI354	-K--A----------Q
AGJ.AU.BFP90	-K--E----------
AGJ.ML.95ML8	-K--E----------R
AGU.CD.Z321	-K--K----------Q--
BF.BR.93BR029.4	----K----------H---R
DF.CD.VI961	-K--A----------R
U.CD.VI1126	-K--S----------R---R
CONSENSUS_CPZ	-k--?----------M
CPZ.CD.CPZANT	EK--T--S----------M
CPZ.GA.CPZGAB	-K------V------R-R-M
CPZ.US.CPZUS	-R--A----------M

GSEELRSLYNTVATLYCVHQ

QUERY	GSEELRSLYNTVATLYCVHQ
CONSENSUS_A	-T------------------
A.KE.Q23-CXC-CG	-T--IK--F-----------
A.SE.SE6594	-T--IK--F-----------
A.SE.SE7253	-T------F----V------
A.SE.SE7535	-T------------------
A.SE.SE8131	-T------------------
A.SE.SE8538	-T---K--------W----
A.SE.SE8891	-T------------------
A.UG.92UG037	-T------------------
A.UG.U455	-T-----------V------
CONSENSUS_B	--------------------
B.AU.AF128998	-----K----A---------
B.-.NL43E9	-----------I-A------
B.AU.MBC18	-----K-V--A--V------
B.AU.MBC200	----IK--------------
B.AU.MBC925	---DF---------------
B.AU.MBCC54	--------------------
B.AU.MBCC98	---D---V------------
B.AU.MBCD36	-----K-------V------
B.CN.RL42	--------F----------L
B.DE.D31	--------F-----------
B.DE.HAN	--------------------
B.ES.89SP061	--------------------
B.FR.HXB2	--------------------
B.GA.OYI	----I---------------
B.GB.CAM1	--------------------
B.GB.MANC	-----K-------V------
B.JP.JH31	-----K--F-----------
B.NL.3202A21	--------F----V------
B.TW.LM49	-----------------I--
B.US.85WCIPR54	--------H----V------
B.US.AD8	-----K--F-----------
B.US.BC	-----K-----I-V------
B.US.DH123	-------------------E
B.US.JRCSF	-----T--------------
B.US.JRFL	--------------------
B.US.MNCG	-----K--------------
B.US.NC7	-----------I--------
B.US.NY5CG	----R---F----V------
B.US.P896	-----K--------------
B.US.RF	-----K----A---------
B.US.SF2	--------------------
B.US.WC001	--------H----V------
B.US.WEAU160	-------------V------
B.US.WR27	--------F-----------
B.US.YU2	--------------------
CONSENSUS_C	-T------?----------?
C.BR.92BR025	-TK--I--H----------E
C.BW.96BW01B22	-T---K------------E
C.BW.96BW0402	-T------F----------K
C.BW.96BW0502	-T----------------A
C.BW.96BW1104	-T--I-------------E
C.BW.96BW1210	-T---K------------E
C.BW.96BW15B03	-T------F----------E
C.BW.96BW1626	-T---K-------V-F---A
C.BW.96BW17A09	-T---K--------------
C.ET.ETH2220	-T---K--F-----------
C.IN.93IN904	-T------H---V------A
C.IN.93IN905	-T------F----------A
C.IN.93IN999	-T------H----------E
C.IN.94IN11246	-T------F----------A
C.IN.95IN21068	-T------F----------A
CONSENSUS_D	--------------------e
D.CD.84ZR085	-------I-----------K
D.CD.ELI	-T-----------------K
D.CD.NDK	----I----------E
D.CD.Z2Z6	--------F----------E
D.UG.94UG1141	----IK------V------E
CONSENSUS_F	-------------V--f---
F.BR.BZ162	-------------V--F---
F.CD.VI174	--------F--IVV--Y---
F.RW.VI69	-------------V--F---
CONSENSUS_F1	-----?--?--?-V--y---
F1.BE.VI850	-----K--F----V--Y---
F1.BR.93BR020.1	-----K-----I-V--Y---
F1.FI.FIN9363	-----------I-V--F---
F1.FR.MP411	--------F----V------
CONSENSUS_F2	-----K--?-??VV--Y---
F2.CM.MP255	-----K----A-VV--Y---
F2.CM.MP257	-----K--F--IVV--Y---
CONSENSUS_G	-T--IK--F-----------
G.BE.DRCBL	-T--IK--F-----------
G.FI.HH8793	-T--IK--F-----------
G.NG.92NG083	-T------F-----------
G.SE.SE6165	-T--IK----A---------
CONSENSUS_H	-T---Q--F----V------
H.BE.VI991	-T-D-Q-----I-V------
H.BE.VI997	-T---x--F---------L-
H.CF.90CF056	-T---K--F-L--V-----R
CONSENSUS_J	-T?-IK--------------
J.SE.SE9173	-T--IK--------------
J.SE.SE9280	-TQ-IK--------------
CONSENSUS_K	-----?--?----?------
K.BE.VI325	-----K--F---V-------
K.CD.EQTB11C	--------F-------W---
K.CM.MP535	----IK-----I-V--F---
N.CM.YBF30	----------AL-V-----S
CONSENSUS_O	--??-?--W-AI?V-W---N
O.CM.ANT70C	--DS-Q--W-AIVV-W---N
O.CM.MVP5180	---D-K--W-AI-V-W---N
CRF01-AE.CF.90CF40	-----K--F--I---W----
CRF01-AE.TH.93TH25	-----K-----I---W----
CRF01-AE.TH.CM240	-L---K--F------W----
CRF01-AE.TH.TH022	--------F------W----
CRF01-AE.TH.TH047	--------F--IV--W----
CRF02_AG.FR.DJ263	-----K-----I---W---K
CRF02_AG.FR.DJ264	-----K-----I---W----
CRF02_AG.NG.IBNG	-----K--F--I---W----
CRF03_AB.RU.KAL15	-----K--------------
CRF04_cpx.CY.94CY0	-----------IT--W----
CRF04_cpx.GR.97PVC	----VK--F--L---W----
CRF04_cpx.GR.97PVM	-----K--F-LI---W----
AC.ET.E3099G	-----K--------------
AC.IN.21301	-T------H----------A
AC.RW.92RW009	-TD-----------------
AC.SE.SE9488	-T--IK--F-----------
AC.ZM.ZAM174-21	-T------F--A-------E
AC.ZM.ZAM184	-T-DI--------V--Y---
AC.ZM.ZAM716-17	-T------F----------A
ACD.SE.SE8603	-T-------------W---K
AD.SE.SE6954	-----K--F----------A
AD.SE.SE7108	-T---K--------------
ADHU.NO.NOGIL3	-----K--F-L--V-W----
ADU.CD.MAL	----IK--------------
AG.NG.G3	-T--IK--F-----------
AG.SE.SE7812	-----K--F--I---W----
AGHU.GA.VI354	-----K--F-----------
AGJ.AU.BFP90	-----K--F-----------
AGJ.ML.95ML8	-----K--------------
AGU.CD.Z321	-T--II--------------
BF.BR.93BR029.4	--------------------
DF.CD.VI961	-------------------E
U.CD.VI1126	--------F---V---W---
CONSENSUS_CPZ	---g----F--l-V-W---s
CPZ.CD.CPZANT	R-P-II--F--ICV-W---K
CPZ.GA.CPZGAB	---G----F--L-V-W-I-S
CPZ.US.CPZUS	---G----F--L-V-W---S

Study Subject ID:02RCH01
Study Subject Clone:
Study Subject HLA:A2,A3,B7,B39,Cw7
Sequence: Known reactive 20Mer0: GELDRWEKIRLRPGGKKKYK p17(11–30)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
A3	A3.1,A*0301,A*0302,A*0304
B39	B*3901,B*3902,B*3903,B*3904,B*3906,B*3908,B*3909,B*3910,B*3912,B*3913,B*39011
B7	B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(2-11)	ELDRWEKIRL	A*0201
(2-11)	ELDRWEKIRL	A*0202
(2-11)	ELDRWEKIRL	A*0204
(2-11)	ELDRWEKIRL	A*0205
(2-11)	ELDRWEKIRL	A*0207
(2-11)	ELDRWEKIRL	A*0214
(10-18)	RLRPGGKKK	A3
(10-17)	RLRPGGKK	A3
(10-19)	RLRPGGKKKY	A3
(4-11)	DRWEKIRL	B*39011
(3-11)	LDRWEKIRL	Cw*0702
(11-19)	LRPGGKKKY	Cw*0702
(4-11)	DRWEKIRL	Cw*0702
(12-19)	RPGGKKKY	Cw*0702
(2-11)	ELDRWEKIRL	Cw*0702
(10-19)	RLRPGGKKKY	Cw*0702

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]

A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
A3	X[LVM]XXXXXX[KYF]
A3	X[LVM]XXXXX[KYF]
A3	X[LVM]XXXXXXX[KYF]
B*39011	X[RH]XXXXXX[L]
B*39011	X[RH]XXXXX[L]
B*39011	X[RH]XXXXXXX[L]
B*3902	X[KQ]XXXXXX[L]
B*3902	X[KQ]XXXXX[L]
B*3902	X[KQ]XXXXXXX[L]
B7	X[P]XXXXXX[LF]
B7	X[P]XXXXX[LF]
B7	X[P]XXXXXXX[LF]
B*0702	X[P]XXXXXX[L]
B*0702	X[P]XXXXX[L]
B*0702	X[P]XXXXXXX[L]
B*0703	X[P]XXXXXX[L]
B*0703	X[P]XXXXX[L]
B*0703	X[P]XXXXXXX[L]
B*0705	X[P]XXXXXX[L]
B*0705	X[P]XXXXX[L]
B*0705	X[P]XXXXXXX[L]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

Study Subject ID:02RCH01
Study Subject Clone:
Study Subject HLA:A2,A3,B7,B39,Cw7
Sequence: Known reactive 20Mer1: GSEELRSLYNTVATLYCVHQ p17(71–90)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
A3	A3.1,A*0301,A*0302,A*0304
B39	B*3901,B*3902,B*3903,B*3904,B*3906,B*3908,B*3909,B*3910,B*3912,B*3913,B*39011
B7	B*07,B*0702,B*0703,B*0704,B*0705,B*0706,B*0707,B*0709,B*0711
Cw7	Cw*0701,Cw*0702,Cw*0704,Cw*0706

Possible Epitopes based on anchor residues

(4-12)	ELRSLYNTV	A*0201
(7-15)	SLYNTVATL	A*0201
(4-12)	ELRSLYNTV	A*0202
(7-15)	SLYNTVATL	A*0202
(7-15)	SLYNTVATL	A*0204
(7-15)	SLYNTVATL	A*0205
(11-18)	TVATLYCV	A*0206
(4-12)	ELRSLYNTV	A*0214
(7-15)	SLYNTVATL	A*0214
(11-18)	TVATLYCV	A*0214
(7-16)	SLYNTVATLY	A3
(1-9)	GSEELRSLY	Cw*0702
(7-15)	SLYNTVATL	Cw*0702
(8-16)	LYNTVATLY	Cw*0702
(1-8)	GSEELRSL	Cw*0702
(2-9)	SEELRSLY	Cw*0702
(8-15)	LYNTVATL	Cw*0702
(9-16)	YNTVATLY	Cw*0702
(6-15)	RSLYNTVATL	Cw*0702
(7-16)	SLYNTVATLY	Cw*0702

Anchor Residues Searched

A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]

A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]
A3	X[LVM]XXXXXX[KYF]
A3	X[LVM]XXXXX[KYF]
A3	X[LVM]XXXXXXX[KYF]
B*39011	X[RH]XXXXXX[L]
B*39011	X[RH]XXXXX[L]
B*39011	X[RH]XXXXXXX[L]
B*3902	X[KQ]XXXXXX[L]
B*3902	X[KQ]XXXXX[L]
B*3902	X[KQ]XXXXXXX[L]
B7	X[P]XXXXXX[LF]
B7	X[P]XXXXX[LF]
B7	X[P]XXXXXXX[LF]
B*0702	X[P]XXXXXX[L]
B*0702	X[P]XXXXX[L]
B*0702	X[P]XXXXXXX[L]
B*0703	X[P]XXXXXX[L]
B*0703	X[P]XXXXX[L]
B*0703	X[P]XXXXXXX[L]
B*0705	X[P]XXXXXX[L]
B*0705	X[P]XXXXX[L]
B*0705	X[P]XXXXXXX[L]
Cw*0702	XXXXXXXX[YFL]
Cw*0702	XXXXXXX[YFL]
Cw*0702	XXXXXXXXX[YFL]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(22–31)	RPGGKKRYKL	RPGGKKKYKL	RPGGKKKYKL	B7	
p17(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
p24(223–231)	GPSHKARVL	GPGHKARVL	GPGHKARVL	B7	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2, A*0202	
RT(308–317)	EILKEPVGHV	EILKEPVHGV	EILKEPVHGV	A*0201	
gp160(121–129)	KLTPLCVSL	KLTPLCVTL	KLTPLCVTL	A2	
gp160(192–200)	KLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2.1	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*07	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B*0702	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7?	
gp160(298–307)	RPNNNTRKSI	RPNYNKRKRI	RPNNNTRKSI	B7	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A*0201	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(311–320)	MGPKRAFYAT	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(369–375)	PEIVTHS	PEIVMHS	PEIVMHS	A2	
gp160(377–387)	NSGGEFFYSNS	NCGGEFFYCNT	NCGGEFFYCNT	A2	
gp160(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLLIV	HHRDLLLIA	RLRDLLLIV	A*0201	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A*0301	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A3	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2.1	
gp160(814–822)	LLNATDIAV	LLNATAIAV	LLNATAIAV	A2	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B*0702	
gp160(843–851)	IPRRIRQGL	IPTRIRQGL	IPRRIRQGL	B7	
Nef(77–85)	RPMTYKAAL	RPMTYKAAV	RPMTYKAAV	B*0702	
Nef(136–145)	PLTFGWCFKL	PLTFGWCYKL	PLTFGWCFKL	A2	

Nef(175–184)	DPEKEVLQWK	DPEREVLEWR	DPEKEVLVWK	B7
Nef(190–198)	AFHHVAREK	AFHHVAREL	AFHHMAREL	A3

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(22–31)	Gag(22–31)	RPGGKKRYKL	HIV-1 infection	human(B7)	[Jin (2000)]
	• This B7 epitope is one of three subdominant CTL responses detected in a long-term non-progressor • A dominant B7 epitope was de£ned using conventional methods, and three additional sub-dominant HLA B7 epitopes were de£ned by £rst using a non-anchor based strategy, EpiMatrix, to identify 2078 possible epitopes in the autologous HIV-1, followed by B7 anchor residue prediction to narrow the set to 55 peptides for experimental testing				
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	• Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL				

Table 2: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(223–231)	p24()	GPSHKARVL	HIV-1 infection	human(B7)	[Goulder (2000a)]
	• The CTL-dominant response was focused on this epitope in a HIV+ Caucasian living in Boston – this epitope did not fall within the three most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

Table 3: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(179–187)	RT()	VIYQYMMDL	HIV-1 exposure	human(A2)	[Rowland-Jones (1998a)]
	• A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D consensus sequences are both VIYQYMMDL				
RT(179–187)	Pol()	VIYQYMMDL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				
RT(308–317)	RT()	EILKEPVGHV	HIV-1 infection	human(A*0201)	[van der Burg (1997), Menendez-Arias (1998)]
	• Recognized by CTL from a long-term survivor, SPIETVPVKL was also recognized • Recognized by CTL from a progressor, EELRQHLLRW and TWETWWTEYW were also recognized				

Table 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(121–129)	gp120(121–129)	KLTPLCVSL	*in vitro* stimulation	human(A2)	[Zarling (1999)]
	• This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL				
gp160(192–200)	gp120(192–199 HXB2R)	KLTSCNTSV	HIV-1 infection	human(A2)	[Brander (1995)]
	• Epitope predicted on HLA binding motif, and studied in the context of inclusion in a synthetic vaccine				
gp160(192–200)	gp120(197–205)	TLTSCNTSV	no CTL shown	human(A2)	[Garboczi (1992)]
	• Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio *et al* 1991				
gp160(192–200)	gp120(199–207)	TLTSCNTSV	peptide immuniza-tion and HIV-1 infection	human(A2.1)	[Brander (1996)]
	• This epitope was recognized by PBMC from 6/14 HIV+ asymptomatic patients • This epitope was used along with pol CTL epitope ALQDSGLEV and a tetanus toxin T helper epitope for a synthetic vaccine • This vaccine failed to induce a CTL response, although a helper response was evident				
gp160(298–307)	gp120(298–307)	RPNNNTRKSI	HIV-1 infection	human(B*07)	[Ferris (1999), Hammond (1995)]
	• The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains an N-linked glycosylation site that is glycosylated in Env • Peptide that had been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) (RPNDNTRKSI) was recognized a 100-fold more ef£ciently than either glycosylated or non-glycosylated RPNNNTRKSI • Position 5 is not involved with HLA B*07 binding, so is probably important for TCR recognition • HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(298–307)	gp120(302–312 HXB2)	RPNNNTRKSI	HIV-1 infection	human(B*0702)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*0702 epitope				
gp160(298–307)	gp120(302–312 HXB2)	RPNNNTRKSI	HIV-1 infection	human(B7)	[Safrit (1994)]
	• CTL from two acute seroconversion cases				
gp160(298–307)	gp120(303–312 IIIB)	RPNNNTRKSI	HIV-1 infection	human(B7?)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RPNNNTRKDI and RPNNNTRKGI, naturally occurring variants, were found in non-transmitting mother – ability to recognize these variants has not yet been determined				
gp160(298–307)	gp120(302–311 Clade B)	RPNNNTRKSI	HIV-1 infection	human(B7)	[Wilson (1998)]
	• The extent of CTL interclade cross-reactivity from CTL isolated from individuals newly infected with B clade virus was studied, and extensive cross-reactivity was observed • Two HLA B7 individuals had CTL response to B_LAI, A_92UG037 and C_92BR025 gp160, but were B clade strain MN non-responders – the authors note that the B7 epitope RPNNNTRKSI is immunodominant, conserved between the LAI and clade A and C strains, but is very divergent in MN (RPNYNKRKRI), and that this epitope might be dominating the speci£city of the response in the HLA B7 individuals				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	CTL line from HIV-donor	human(A*0201)	[Alexander-Miller (1996)]
	• This immunogenic peptide does not have the known binding motif for A2.1 • The same optimal peptide for this human HLA-A2.1 epitope was observed for a murine H-2 D^d epitope				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 IIIB and boosted with puri£ed gp160 • Lysis only occurs with IIIB P18 peptide pulsed onto autologous targets; MN, RF, SIMI P18 peptides fail to stimulate CTL • Restimulating immune cells from gp160 IIIB vaccinees with MN, RF, or SIMI P18 did not enhance the MN, RF, or SIMI speci£c CTL response				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(311–320)	gp160(318–327 SIMI)	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 SIMI and boosted with puri£ed recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-speci£c CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYTT) and the P18 RF peptide (KGPGRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPGRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a signi£cantly broader speci£city when stimulated with P18 MN or P18RF peptides, but not P18 IIIB			
gp160(369–375)	gp120(374–380 BRU)	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]
	• De£ned through blocking CTL activity, and Env deletions				
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
	• Peptides recognized by class I restricted CTL can bind to class II				
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
	• This epitope is processed by a TAP1/2 dependent mechanism				
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
	• Studied in the context of HLA-A2 peptide binding				
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
	• CTL responses in six patients to four Env epitopes were studied: D2: LLNATAIAV, 5.3: RLRDLLLIV, D1: KLTPLCVTL, and 4.3: QMHEDIISL – all have A2 anchor residues • The C terminal epitopes (D2 and 5.3) were highly variable and the variability was considered responsible for limited CTL response, while D1 and 4.3, N-terminal epitopes, were much more conserved and gave evidence of high levels of CTL response *in vitro* • Peptides 5.3 and D2 bound to HLA A*0201 with low af£nity and were variable, particularly D2;			
gp160(770–780)	gp41(768–778 NL43)	RLRDLLLIVTR	HIV-1 infection	human(A*0301)	[Takahashi (1991)]
	• CD8+ T cell clone				
gp160(770–780)	gp41(768–778 NL43)	RLRDLLLIVTR	HIV-1 infection	human(A3)	[Cao (1997)]
	• The consensus peptide of clade B is RLRDLLLIVTR • The consensus peptide of clades A, C and E is RLRDFILIVTR and it is less reactive • The consensus peptide of clade D is SLRDLLLIVTR and it is less reactive				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(813–822)	gp41(814–823 LAI)	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823 • Noted to be A*0201 in Brander *et al*., 1999 database				
gp160(813–822)	gp41(814–823)	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLLNATDIAV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, and 3 of these had a detectable CTL response – the other two had either the sequence SLFNAIDIAV or SLLNTTDIVV and no detectable CTL response • CTL demonstrated against peptide-coated target, epitope is naturally processed and enhancible with vaccine				
gp160(813–822)	Env(814–823 Clade B)	SLLNATDIAV	HIV-1 MN rgp160	human(A2.1)	[Kundu (1998a)]
	• Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and £fty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identi£ed in gp160, of which 25 had a high or intermediate binding af£nity • Eleven peptides were studied that had high HLA-A2 binding af£nity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses • CTL to overlapping peptides in this region gave a positive response in the greatest number of patients • ALTERNATIVE EPITOPES: LLNATDIAV and LLNATDIAVA – CTL were induced by vaccine in those that had the sequence SLLNATAIAVA in their own infection, but not in those with: NLLNTIAIAVA or NLFNTTAIAVA or SLLNATAITVA				
gp160(814–822)	gp41(815–823 LAI)	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823				
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B*0702)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*0702 epitope				
gp160(843–851)	gp41(848–856 LAI)	IPRRIRQGL		human(B7)	[Brander & Walker(1995)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study				

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(77–85)	Nef(77–85 LAI)	RPMTYKAAL	HIV-1 infection	human(B*0702)	[Bauer (1997)]
	• Structural constraints on the Nef protein may prevent escape • Noted in Brander 1999, this database, to be B*0702				
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL				
Nef(175–184)	Nef(175–184)	DPEKEVLQWK	HIV-1 infection	human(B7)	[Jin (2000)]
	• This a B7 epitope, a subdominant CTL response, was de£ned by an un-conventional approach used to predict epitopes in an HLA B7+ long-term non-progressor • Three additional sub-dominant HLA B7 epitopes were de£ned using EpiMatrix, a non-anchor based strategy for de£ning potential epitopes, which highlighted 2078 possible epitopes in the autologous HIV-1 derived from the study subject, followed by B7 anchor residue prediction which narrowed the set to 55 peptides, three of which could serve as functional CTL epitopes				
Nef(190–198)	Nef(190–198 LAI)	AFHHVAREK	HIV-1 infection	human(A3)	[Hadida (1995)]
	• Naturally occurring L to K anchor substitution abrogates A2 binding, but permits HLA-A3 binding				

Table 6: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK		human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*0301 epitope				
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • KIRLRPGGR and RIRLRPGGR, naturally occurring variants, were found in mother, and are escape mutants				
p17(18–26)	p17(18–26)	KIRLRPGGK	*in vitro* stimulation	human(A3)	[Zarling (1999)]
	• This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL			
p17(18–26)	Gag(18–26)	KIRLRPGGK	HIV-1 infection	human(A3)	[Brodie (1999)]
	• The ability of CTL effector cells was studied by expanding autologous HIV-1 Gag-speci£c CTL *in vitro*, and adoptive transfer • The transferred CTLs migrated to the lymph nodes and transiently reduced circulating productively-infected CD4+ T cells, showing that CTL move to appropriate target sites and mediate anti-viral effects				
p17(18–26)	(18–26)	KIRLRPGGK	HIV infection	human(A3)	[Brodie (2000)]
	• Study tracks and quanti£es *in vivo* migration of neo-marked CD8 HIV-speci£c CTL • Adoptively transferred gene-marked HIV-speci£c CTL homed to speci£c lymph node sites, colocalizing within the parafollicular regions of the lymph node adjacent to cells expressing HIV tat-fusion transcripts, indicative of viral replication • The CTL clones expressed CCR5 and localized among HIV-1 infected cells expressing MIP-1alpha and MIP-1beta, CC-chemokines produced at sites of viral replication, suggesting a possible homing mechanism • This study provides a methodology for tracking and studying antigen speci£c CTL *in vivo*				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	SJL/J HLA transgenic mice(A3)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • KIRLRPGGR and RIRLRPGGR were escape mutants • This epitope was recognized and many escape mutants were detected in an HLA A3 transmitting mother, and was recognized but invariant in an HLA A3 non-transmitting mother				
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
	• Identical twin hemophiliac brothers were both infected with the same batch of factor VIII. One had a response to this epitope, the other did not. [Goulder (1997b)] is a review of immune escape that summarizes this study.				
p17(18–26)	p17()	KIRLRPGGK	HIV-1 exposed seronegative	human(A3)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women				
p17(18–26)	p17()	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 7/10 that had a dominant response to this epitope were A3, and 5/7 targeted RLRPGGKKK while 2/7 targeted KIRLRPGGK (this tally comes from the tables, not the text of the paper) • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPGGKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	()	KIRLRPGGK	HIV-1 infection	human(B*0301)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p17(18–27)	p17(18–27 LAI)	KIRLRPGGKK		human(B27)	[Brander & Walker(1996)]
	• D. Lewinsohn, pers. comm.				
p17(18–27)	p17(18–27)	KIRLRPGGKK	HIV-1 infection	human(B27)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(19–27)	p17(19–27 JRCSF)	IRLRPGGKK	HIV-1 infection	scid-hu mouse(B*2705)	[Brander & Goulder(2001)]
	Noted by Brander to be B*2705 (Pers. Comm. D. Lewinsohn)				
p17(19–27)	p17(19–27 LAI)	IRLRPGGKK		human(B27)	[Brander & Walker(1996)]
p17(19–27)	p17(19–27 JRCSF)	IRLRPGGKK	HIV-1 infection	scid-hu mouse(B27)	[McKinney (1999)]
	• Epitope-speci£c CTL were infused in infected human PBL-SCID mice, and transient decreases in viral load were observed, however virus was not eradicated and the HIV-speci£c CTL rapidly disappeared • No escape mutants were observed • Control CTL were long lived in both infected and uninfected mice, showing the rapid loss of CTL was due to target interaction				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(19–27)	p17()	IRLRPGGKK	HIV-1 infection	human(B27)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 2/3 individuals that were B27+ had a dominant response to this epitope • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human()	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • Three of the four individuals that responded to SLYNTVATL recognized HIV epitopes, and one individual who was A*0201, A31 and B51 and B58w4 recognized this epitope (previously described as HLA A3.1), as well as one other				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A*03)	[Goulder (1997b), Goulder (1997a)]
	• Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • One had a response to gag A3 epitope RLRPGGKKK, the other non-responder carried the sequence RLRPGGKKC • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*0301				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–28)	p17()	RLRPGGKKK	HIV-1 infection	human(A*0301)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (2000b)]
	• Two clonal CTL responses were generated in donor 021-BMC (HLA A3/3001, B42/-, Cw17/-) against different optimal versions of this epitope, one nine amino acids long, one ten • A previously described optimal A3 epitope overlapping this region, KIRLRPGGK, was not recognized by CTL from 021-BMC				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (1997c)]
	• A control CTL line that reacts with this peptide was included in the study				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Cao (1997)]
	• The consensus peptide of A, B, and D clade viruses is RLRPGGKKK • The consensus peptide of C clade viruses is RLRPGGKKH and is equally reactive				
p17(20–28)	p17()	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 7/10 that had a dominant response to this epitope were A3, and 5/7 targeted RLRPGGKKK while 2/7 targeted KIRLRPGGK (this tally comes from the tables, not the text of the paper which stated 6/7 RLRPGGKKK) • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPGGKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY	HIV-1 infection	human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0301 epitope				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A3)	[Goulder (2000b)]
	• Two clonal CTL responses were generated in donor 021-BMC (HLA A3/3001, B42/-, Cw17/-) against different optimal versions of this epitope, one nine amino acids long, one ten • A previously described optimal A3 epitope overlapping this region, KIRLRPGGK, was not recognized by CTL from 021-BMC				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Brander & Walker(1995)]
	• Unpublished, C. Jassoy and Beatrice Culman, pers. comm.				
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Wilkens & Ruhl(1999)]
	• Pers. comm., B. Wilkens and D. Ruhl				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A30,A3.1)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was A30, and one was A3, and both responded to RLRPGGKKKY • The A2+ A3 individual also reacted with two other A3.1 epitopes				
p17(20–29)	p17(20–29 IIIB)	RLRPGGKKKY	HIV-1 infection	human(B42)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RLRPGGKKRY, a naturally occurring variant, was found in non-transmitting mother and is recognized • Binds HLA-A3 and Bw62 as well				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV infection	human(B62)	[Brodie (2000)]
	• Study tracks and quanti£es *in vivo* migration of neo-marked CD8 HIV-speci£c CTL • Adoptively transferred gene-marked HIV-speci£c CTL homed to speci£c lymph node sites, colocalizing within the parafollicular regions of the lymph node adjacent to cells expressing HIV tat-fusion transcripts, indicative of viral replication • The CTL clones expressed CCR5 and localized among HIV-1 infected cells expressing MIP-1alpha and MIP-1beta, CC-chemokines produced at sites of viral replication, suggesting a possible homing mechanism • This study provides a methodology for tracking and studying antigen speci£c CTL *in vivo*				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY		human(Bw62)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also P. Johnson, pers. comm.				
p17(20–30)	p17()	RLRPGGKKKYK	HIV-1 infection	human()	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – the dominant response in a Haitian immigrant living in Boston who was HLA A24/29 B7/B44 Cw6/7 was to this epitope, although the restricting element was not determined • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

Table 7: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK		human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*0301 epitope				
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • KIRLRPGGR and RIRLRPGGR, naturally occurring variants, were found in mother, and are escape mutants				
p17(18–26)	p17(18–26)	KIRLRPGGK	*in vitro* stimulation	human(A3)	[Zarling (1999)]
	• This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL			
p17(18–26)	Gag(18–26)	KIRLRPGGK	HIV-1 infection	human(A3)	[Brodie (1999)]
	• The ability of CTL effector cells was studied by expanding autologous HIV-1 Gag-speci£c CTL *in vitro*, and adoptive transfer • The transferred CTLs migrated to the lymph nodes and transiently reduced circulating productively-infected CD4+ T cells, showing that CTL move to appropriate target sites and mediate anti-viral effects				
p17(18–26)	(18–26)	KIRLRPGGK	HIV infection	human(A3)	[Brodie (2000)]
	• Study tracks and quanti£es *in vivo* migration of neo-marked CD8 HIV-speci£c CTL • Adoptively transferred gene-marked HIV-speci£c CTL homed to speci£c lymph node sites, colocalizing within the parafollicular regions of the lymph node adjacent to cells expressing HIV tat-fusion transcripts, indicative of viral replication • The CTL clones expressed CCR5 and localized among HIV-1 infected cells expressing MIP-1alpha and MIP-1beta, CC-chemokines produced at sites of viral replication, suggesting a possible homing mechanism • This study provides a methodology for tracking and studying antigen speci£c CTL *in vivo*				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	SJL/J HLA transgenic mice(A3)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • KIRLRPGGR and RIRLRPGGR were escape mutants • This epitope was recognized and many escape mutants were detected in an HLA A3 transmitting mother, and was recognized but invariant in an HLA A3 non-transmitting mother				
p17(18–26)	p17(18–26 IIIB)	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder (1997b), Goulder (1997a)]
	• Identical twin hemophiliac brothers were both infected with the same batch of factor VIII. One had a response to this epitope, the other did not. [Goulder (1997b)] is a review of immune escape that summarizes this study.				
p17(18–26)	p17()	KIRLRPGGK	HIV-1 exposed seronegative	human(A3)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women				
p17(18–26)	p17()	KIRLRPGGK	HIV-1 infection	human(A3)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 7/10 that had a dominant response to this epitope were A3, and 5/7 targeted RLRPGGKKK while 2/7 targeted KIRLRPGGK (this tally comes from the tables, not the text of the paper) • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPGGKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(18–26)	()	KIRLRPGGK	HIV-1 infection	human(B*0301)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p17(18–27)	p17(18–27 LAI)	KIRLRPGGKK		human(B27)	[Brander & Walker(1996)]
	• D. Lewinsohn, pers. comm.				
p17(18–27)	p17(18–27)	KIRLRPGGKK	HIV-1 infection	human(B27)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(19–27)	p17(19–27 JRCSF)	IRLRPGGKK	HIV-1 infection	scid-hu mouse(B*2705)	[Brander & Goulder(2001)]
	Noted by Brander to be B*2705 (Pers. Comm. D. Lewinsohn)				
p17(19–27)	p17(19–27 LAI)	IRLRPGGKK		human(B27)	[Brander & Walker(1996)]
p17(19–27)	p17(19–27 JRCSF)	IRLRPGGKK	HIV-1 infection	scid-hu mouse(B27)	[McKinney (1999)]
	• Epitope-speci£c CTL were infused in infected human PBL-SCID mice, and transient decreases in viral load were observed, however virus was not eradicated and the HIV-speci£c CTL rapidly disappeared • No escape mutants were observed • Control CTL were long lived in both infected and uninfected mice, showing the rapid loss of CTL was due to target interaction				

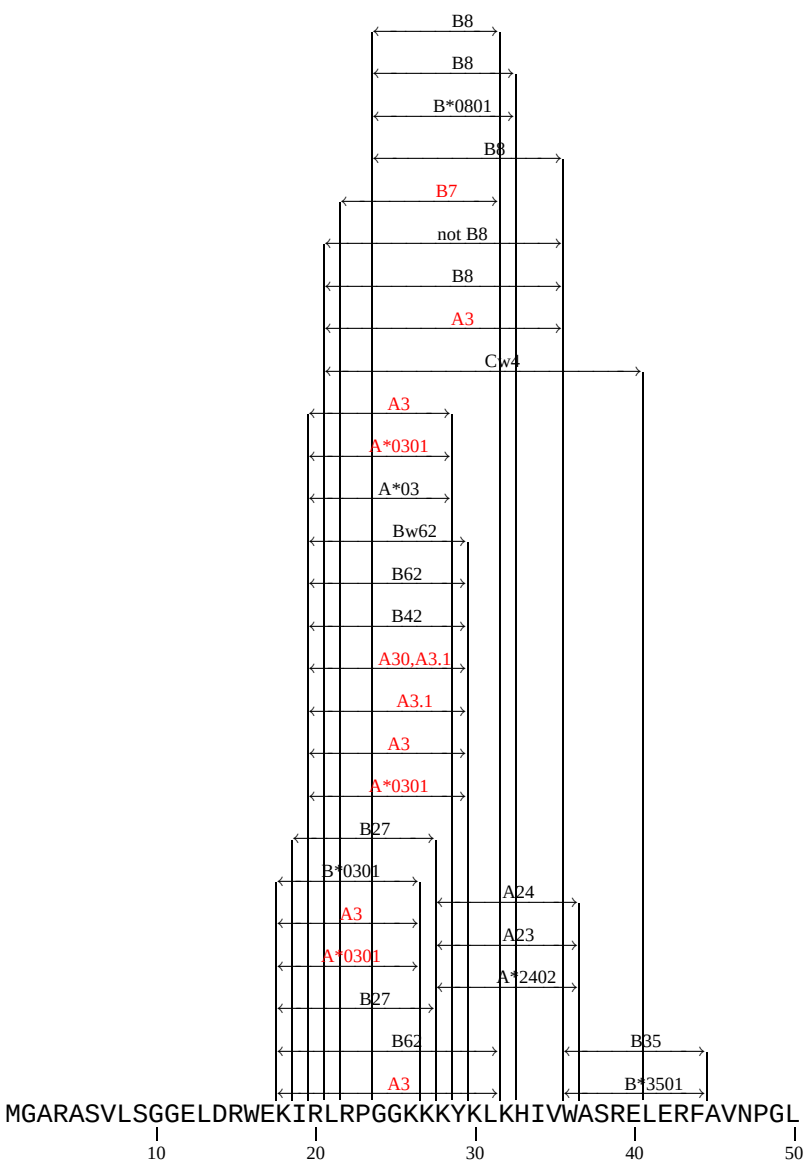
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(19–27)	p17()	IRLRPGGKK	HIV-1 infection	human(B27)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 2/3 individuals that were B27+ had a dominant response to this epitope • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human()	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • Three of the four individuals that responded to SLYNTVATL recognized HIV epitopes, and one individual who was A*0201, A31 and B51 and B58w4 recognized this epitope (previously described as HLA A3.1), as well as one other				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A*03)	[Goulder (1997b), Goulder (1997a)]
	• Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • One had a response to gag A3 epitope RLRPGGKKK, the other non-responder carried the sequence RLRPGGKKC • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes that this is an A*0301				

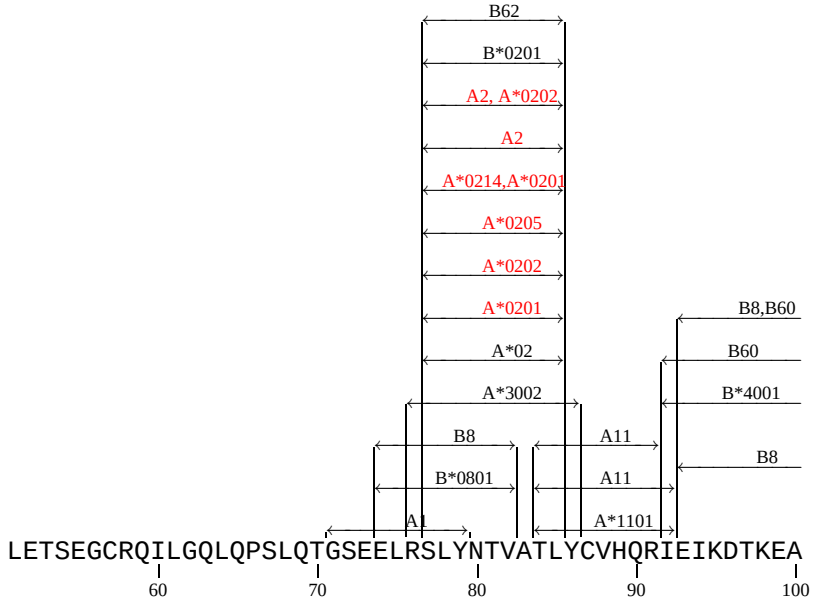
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–28)	p17()	RLRPGGKKK	HIV-1 infection	human(A*0301)	[Wilson (2000)]
	• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (2000b)]
	• Two clonal CTL responses were generated in donor 021-BMC (HLA A3/3001, B42/-, Cw17/-) against different optimal versions of this epitope, one nine amino acids long, one ten • A previously described optimal A3 epitope overlapping this region, KIRLRPGGK, was not recognized by CTL from 021-BMC				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (1997c)]
	• A control CTL line that reacts with this peptide was included in the study				
p17(20–28)	p17(20–28)	RLRPGGKKK	HIV-1 infection	human(A3)	[Cao (1997)]
	• The consensus peptide of A, B, and D clade viruses is RLRPGGKKK • The consensus peptide of C clade viruses is RLRPGGKKH and is equally reactive				
p17(20–28)	p17()	RLRPGGKKK	HIV-1 infection	human(A3)	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – 7/10 that had a dominant response to this epitope were A3, and 5/7 targeted RLRPGGKKK while 2/7 targeted KIRLRPGGK (this tally comes from the tables, not the text of the paper which stated 6/7 RLRPGGKKK) • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPGGKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

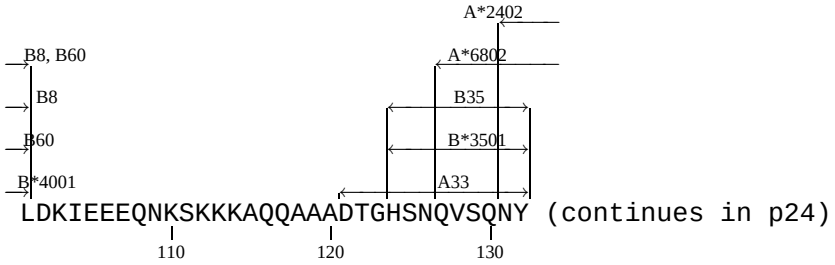
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY	HIV-1 infection	human(A*0301)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0301 epitope				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A3)	[Goulder (2000b)]
	• Two clonal CTL responses were generated in donor 021-BMC (HLA A3/3001, B42/-, Cw17/-) against different optimal versions of this epitope, one nine amino acids long, one ten • A previously described optimal A3 epitope overlapping this region, KIRLRPGGK, was not recognized by CTL from 021-BMC				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Brander & Walker(1995)]
	• Unpublished, C. Jassoy and Beatrice Culman, pers. comm.				
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY	HIV-1 infection	human(A3.1)	[Wilkens & Ruhl(1999)]
	• Pers. comm., B. Wilkens and D. Ruhl				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV-1 infection	human(A30,A3.1)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • 1/11 of the A2+ individuals was A30, and one was A3, and both responded to RLRPGGKKKY • The A2+ A3 individual also reacted with two other A3.1 epitopes				
p17(20–29)	p17(20–29 IIIB)	RLRPGGKKKY	HIV-1 infection	human(B42)	[Wilson (1996)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • RLRPGGKKRY, a naturally occurring variant, was found in non-transmitting mother and is recognized • Binds HLA-A3 and Bw62 as well				
p17(20–29)	p17(20–29)	RLRPGGKKKY	HIV infection	human(B62)	[Brodie (2000)]
	• Study tracks and quanti£es *in vivo* migration of neo-marked CD8 HIV-speci£c CTL • Adoptively transferred gene-marked HIV-speci£c CTL homed to speci£c lymph node sites, colocalizing within the parafollicular regions of the lymph node adjacent to cells expressing HIV tat-fusion transcripts, indicative of viral replication • The CTL clones expressed CCR5 and localized among HIV-1 infected cells expressing MIP-1alpha and MIP-1beta, CC-chemokines produced at sites of viral replication, suggesting a possible homing mechanism • This study provides a methodology for tracking and studying antigen speci£c CTL *in vivo*				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(20–29)	p17(20–29 LAI)	RLRPGGKKKY		human(Bw62)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes • Also P. Johnson, pers. comm.				
p17(20–30)	p17()	RLRPGGKKKYK	HIV-1 infection	human()	[Goulder (2000a)]
	• WEKIRLRPGGKKKYKLK was the target of the dominant response in Caucasoids (38%) more frequently than non-Caucasoids (12%) – the dominant response in a Haitian immigrant living in Boston who was HLA A24/29 B7/B44 Cw6/7 was to this epitope, although the restricting element was not determined • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			

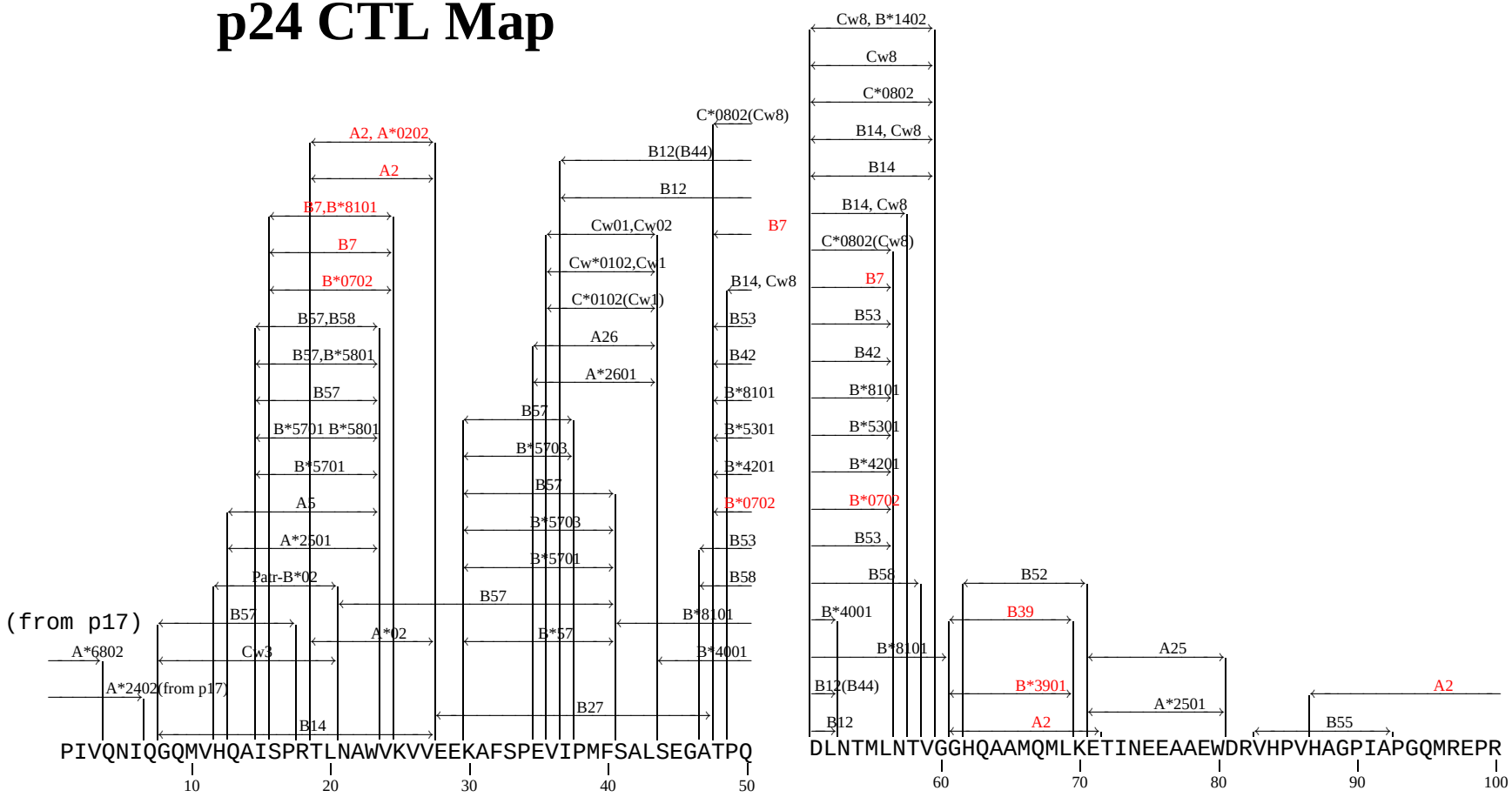
p17 CTL Map

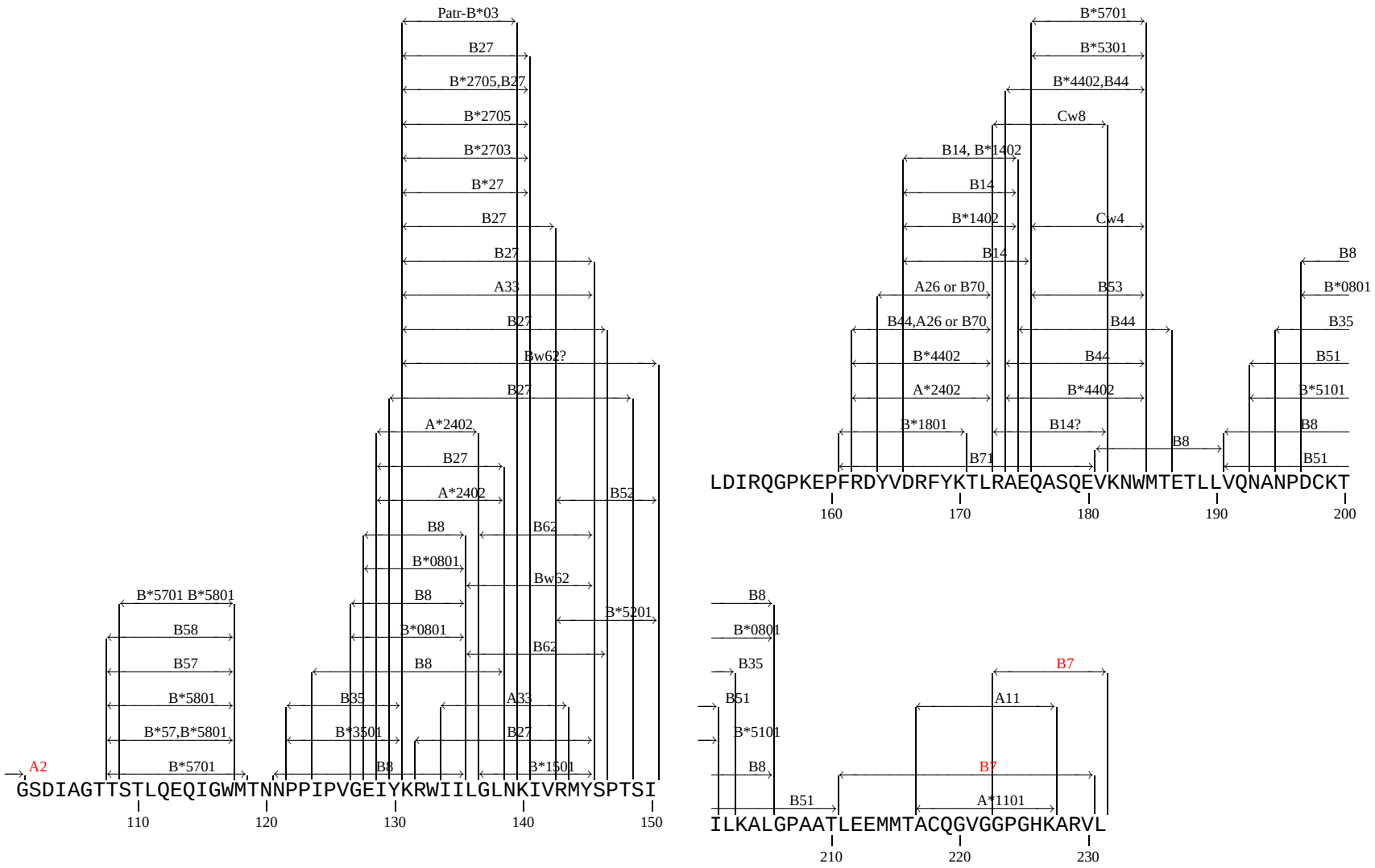

B8
B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
MGARASVLSGGELDRWEKIRLRPGGKKKYKLKHIVWASRELERFAVNPGL
10
20
30
40
50


B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
B60
B*4001
B8
LETSEGCRQILGQLQPSLQTGSEELRSLYNTVATLYCVHQRIEIKDTKEA
60
70
80
90
100

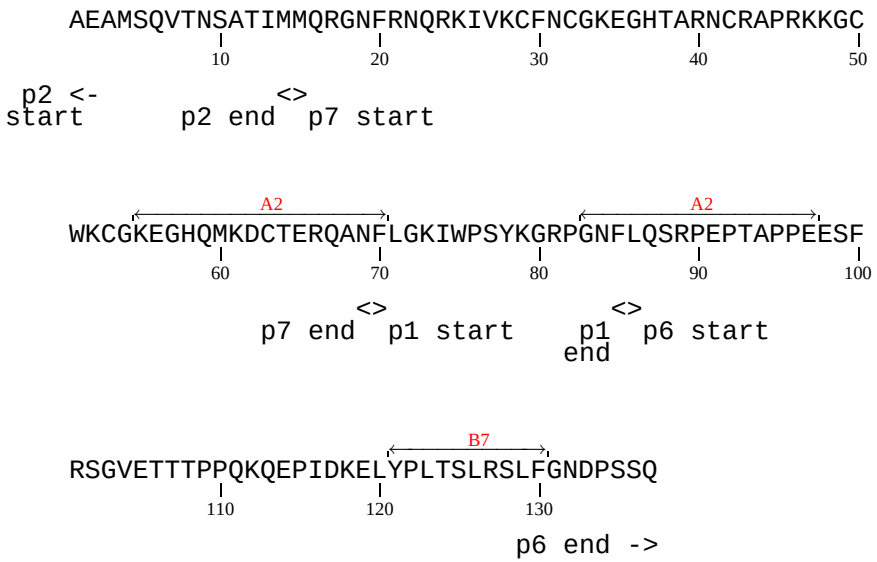

A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
LDKIEEEQNKSKKKAQQAAADTGHSNQVSQNY (continues in p24)
110
120
130

p24 CTL Map

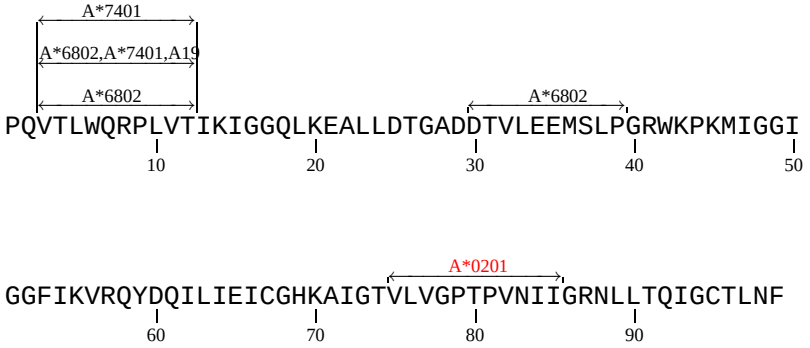

(from p17)
A*6802
A*2402(from p17)
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*57
B27
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
PIVQNIQGQMVHQAISPRTLNAWVKVVEEKAFSPEVIPMFSALSEGATPQ
10
20
30
40
50
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
B12(B44)
B12
B52
B39
B*3901
A2
A25
A*2501
A2
B55
DLNTMLNTVGGHQAAMQMLKETINEEAAEWDRVHPVHAGPIAPGQMREPR
60
70
80
90
100


Patr-B*03
B27
B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5201
B62
B*5701 B*5801
B58
B57
B*5801
B*57,B*5801
B*5701
A2
B8
B*0801
B8
B*0801
B35
B51
B*5101
B8
B51
B35
A33
B*3501
B27
B8
B*1501
GSDIAGTTSTLQEQIGWMTNNPPIPVGEIYKRWIILGLNKIVRMYSPTSI
110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
A26 or B70
B53
B44,A26 or B70
B44
B*4402
B44
A*2402
B*4402
B*1801
B14?
B71
B8
B8
B*0801
B35
B51
B*5101
B8
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B7
A11
B7
A*1101
ILKALGPAATLEEMMTACQGVGGPGHKARVL
210
220
230

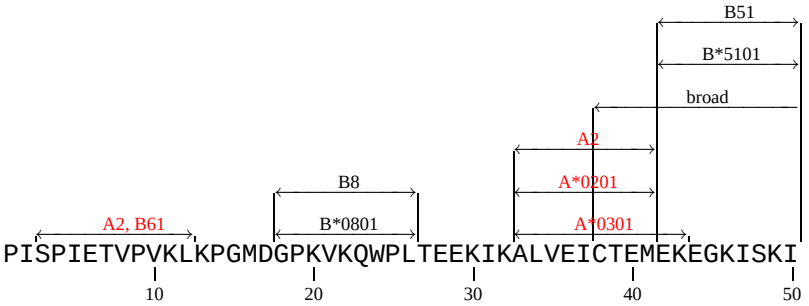
p2p7p1p6 CTL Map



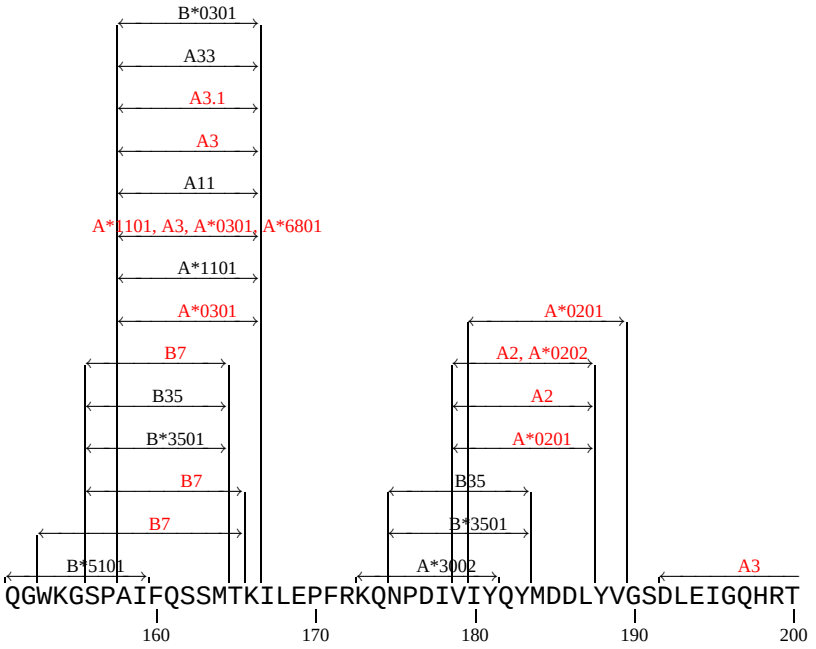
Protease CTL Map

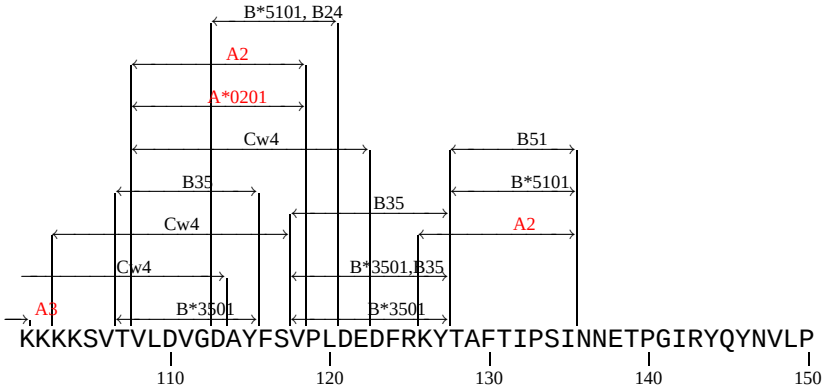


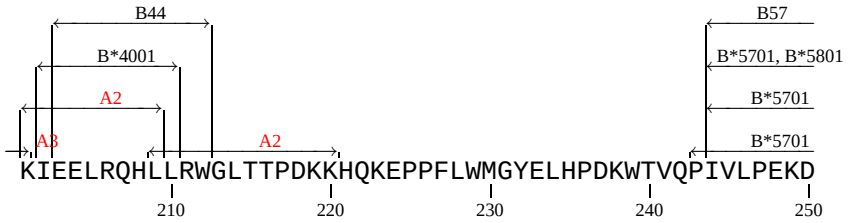
RT CTL Map

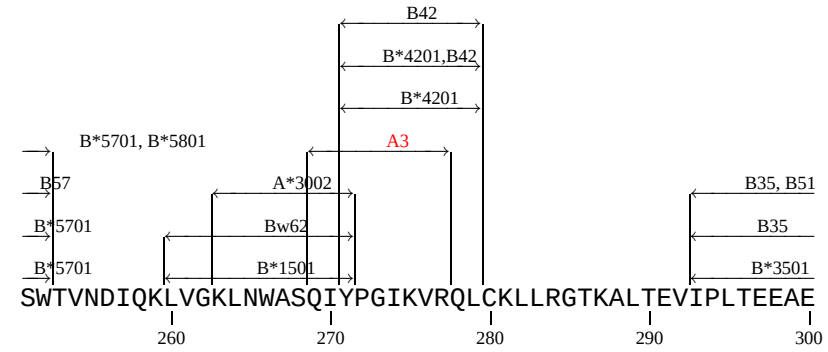

B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50

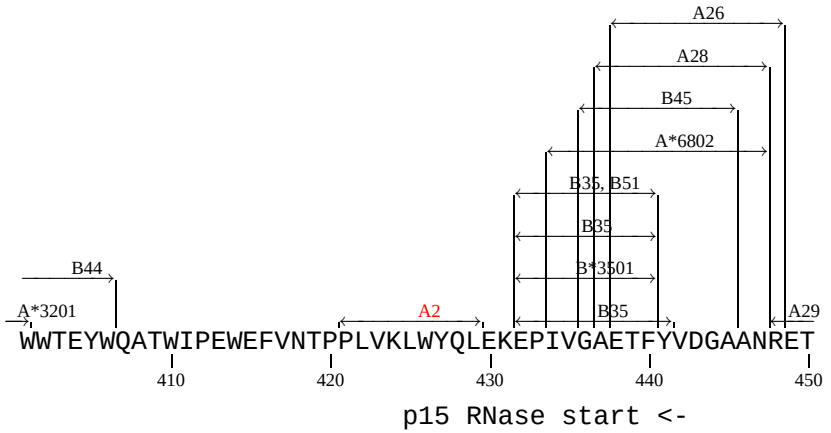

Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200

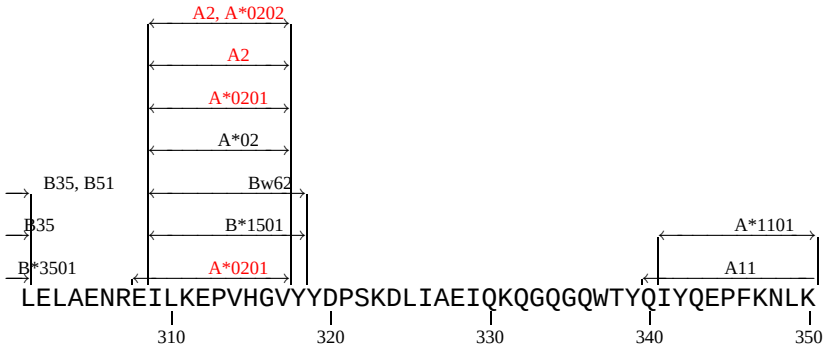

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

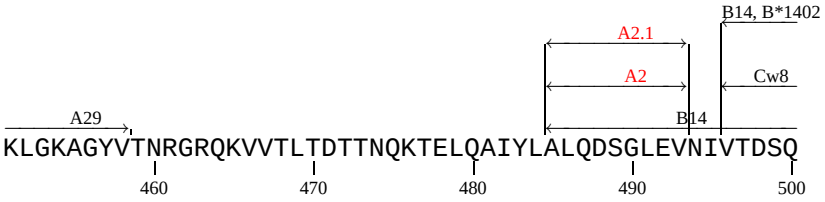

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

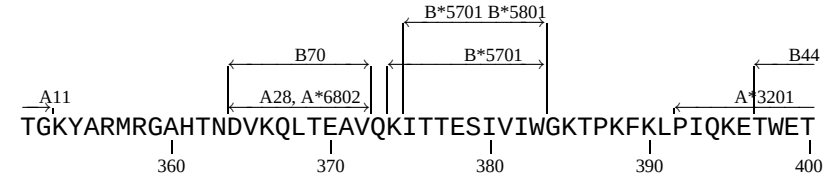


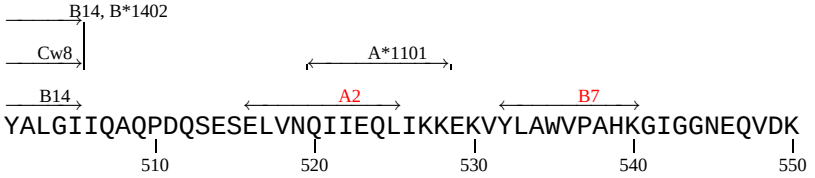


p15 RNase start <-





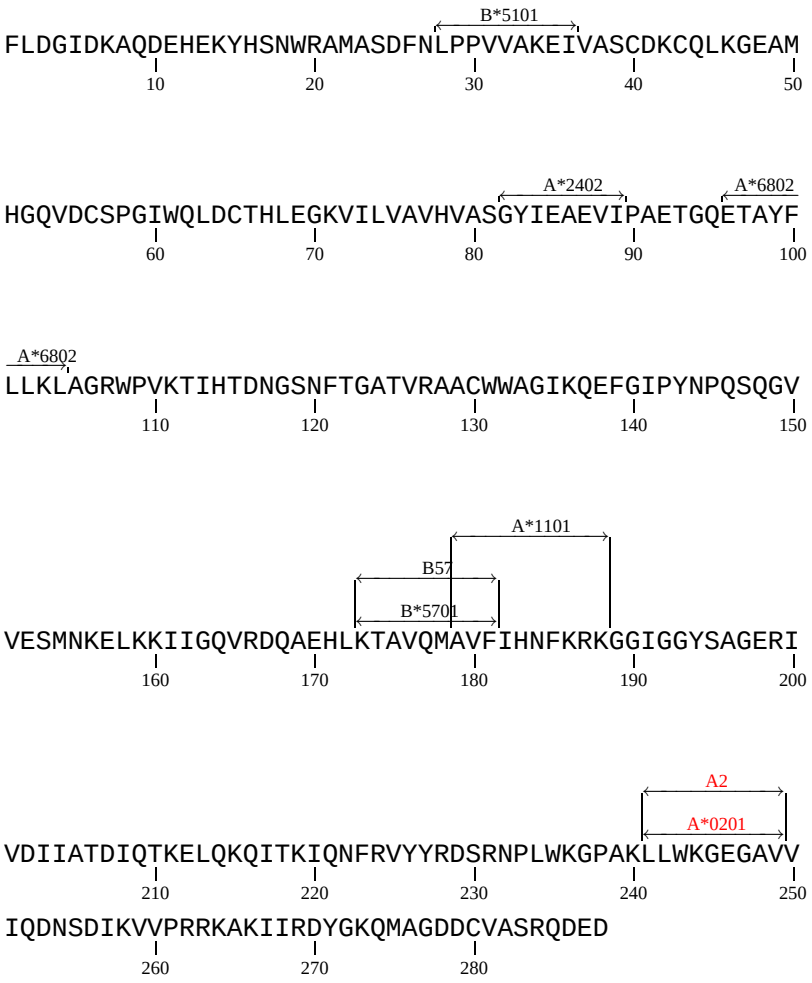




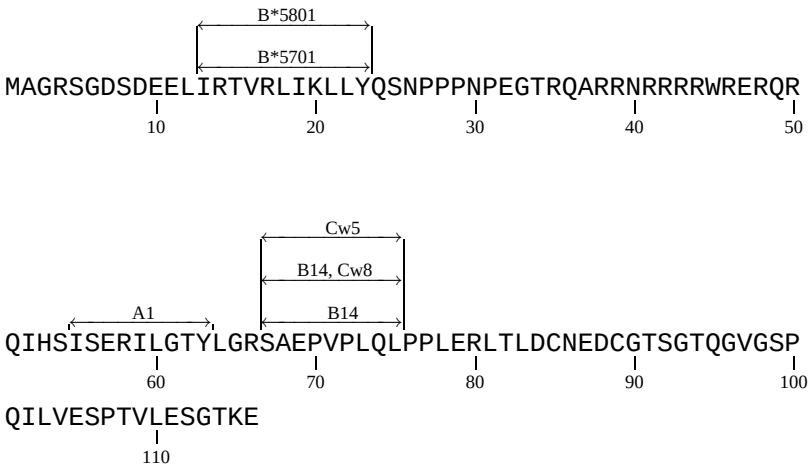
LVSAGIRKVL

-> p15 RNase end

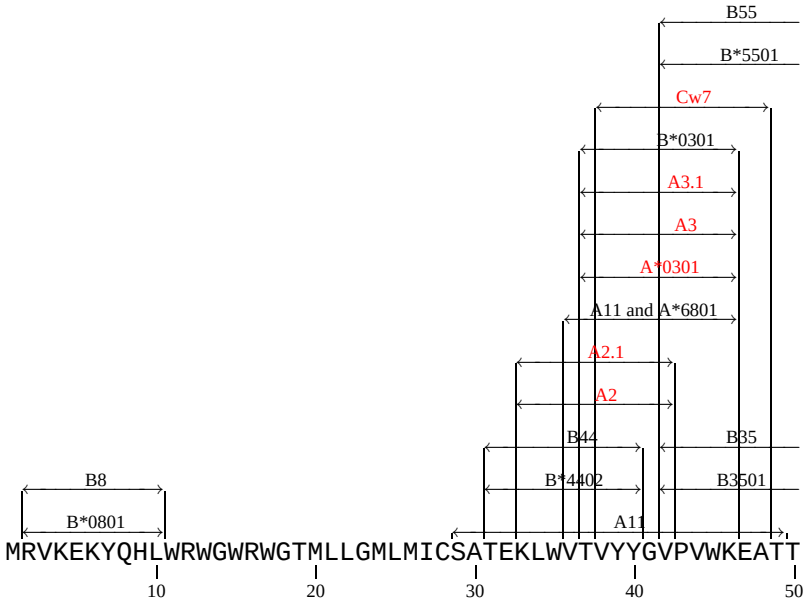
Integrase CTL Map



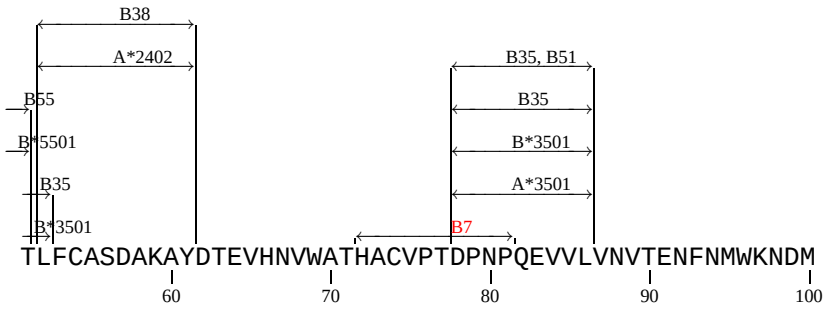
Rev CTL Map

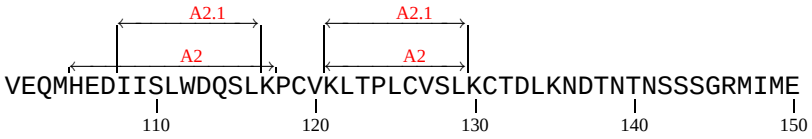


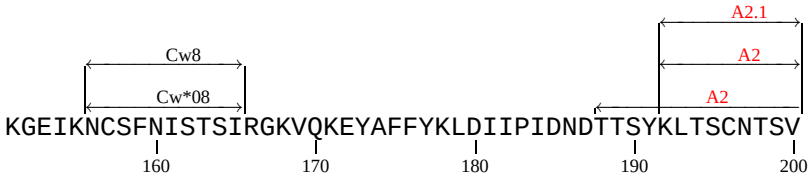
gp160 CTL Map

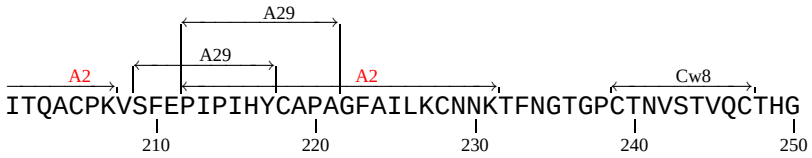


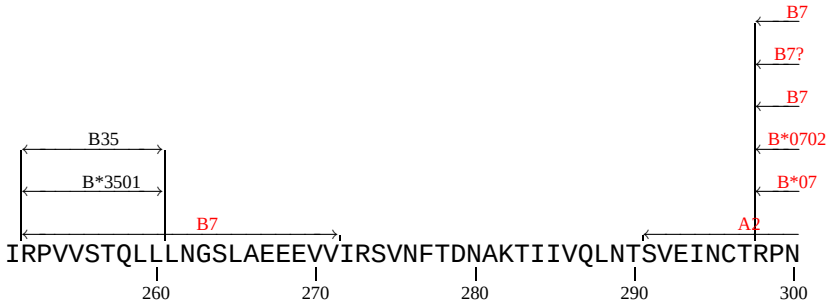
<- gp120 start

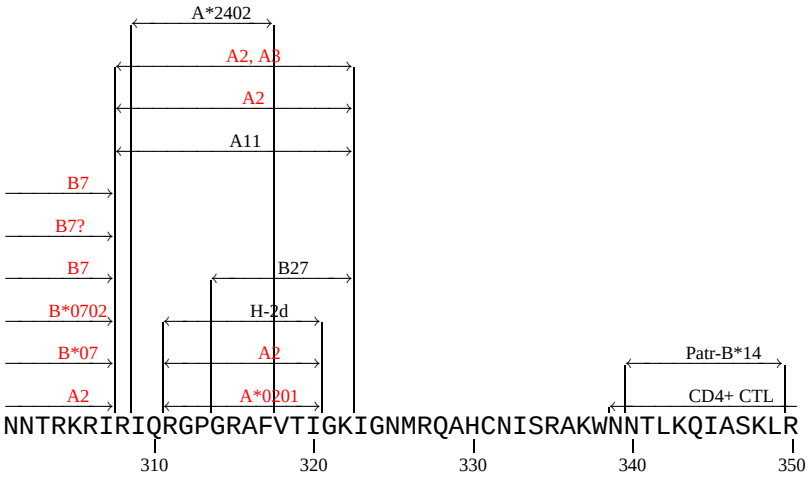


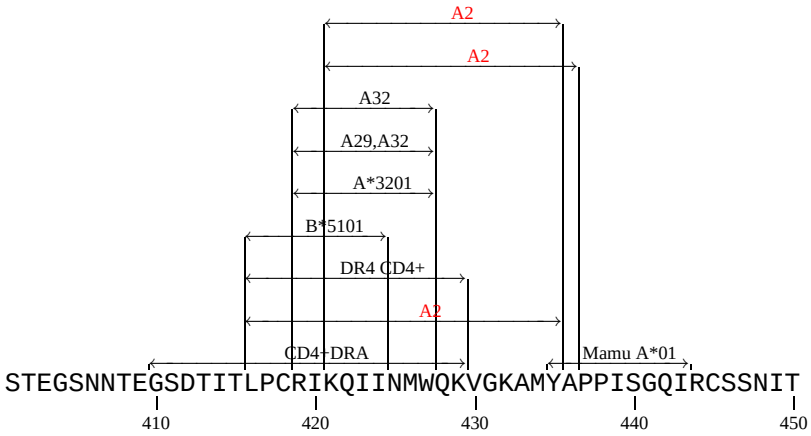


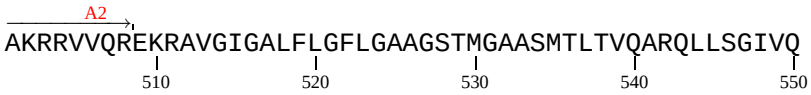




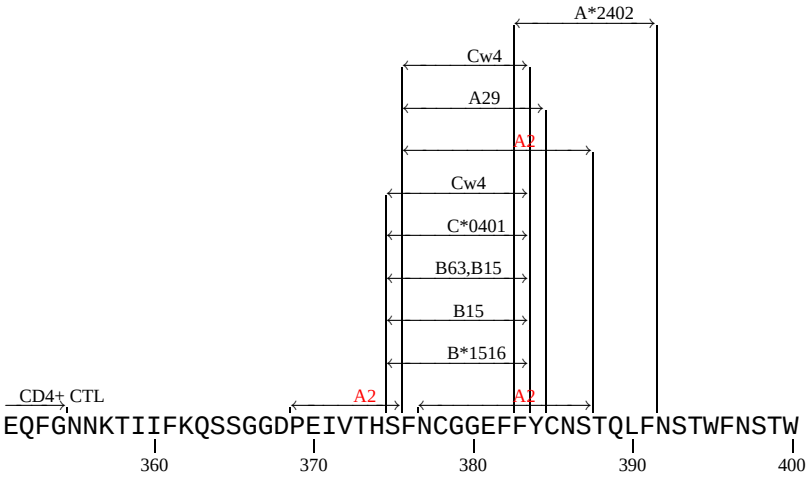


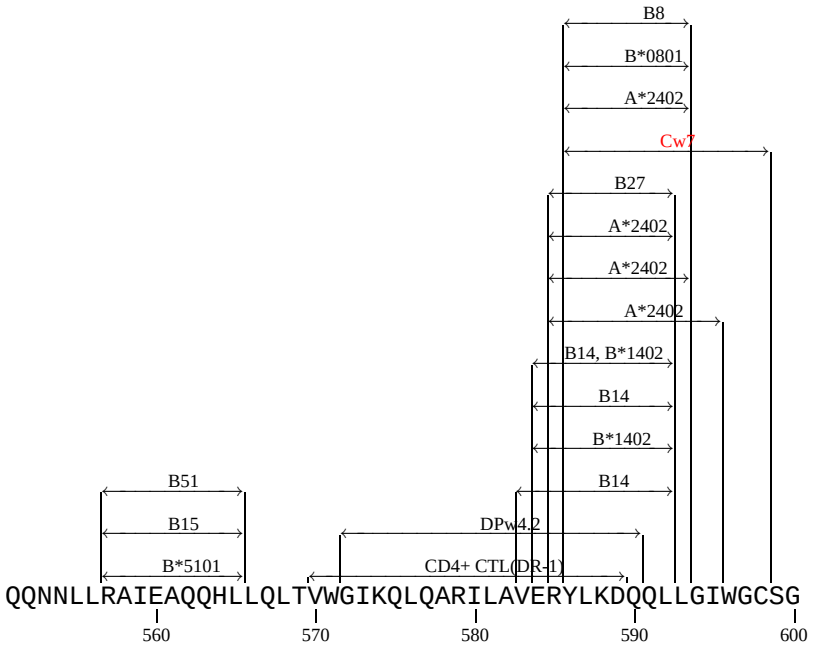


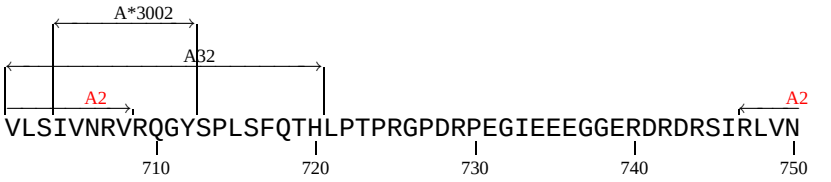


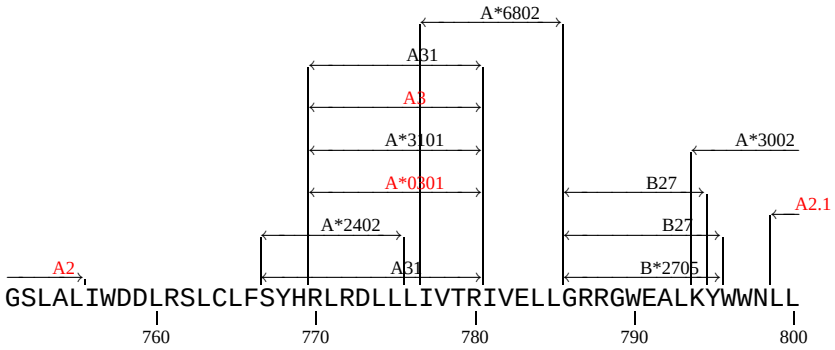


gp120 end <> gp41 start



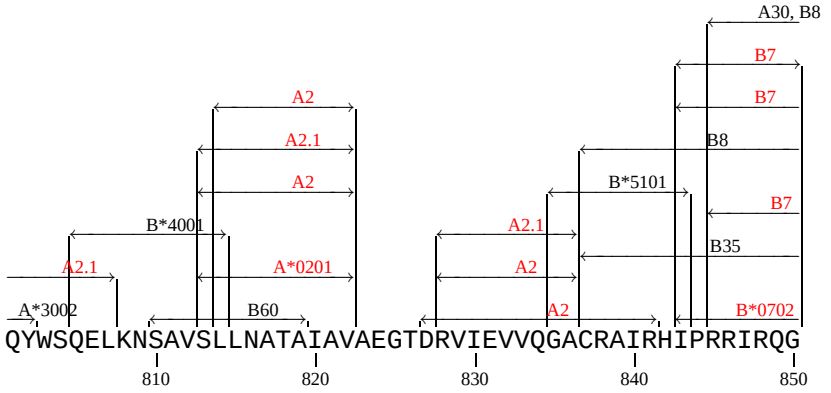

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600

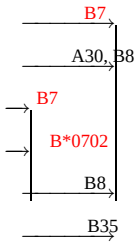

A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A31
A*3002
B27
B27
B*2705
A2.1
A2
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650


A2.1
A*2402
A2.1
A2
NQQEKNEQELLELDKWASLWNWFNITNWLWYIKLFIMIVGGLVGLRIVFA
660
670
680
690
700

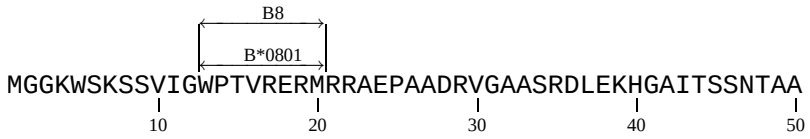

A30, B8
B7
A2
B7
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A2.1
A*0201
A2
A*3002
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
810
820
830
840
850

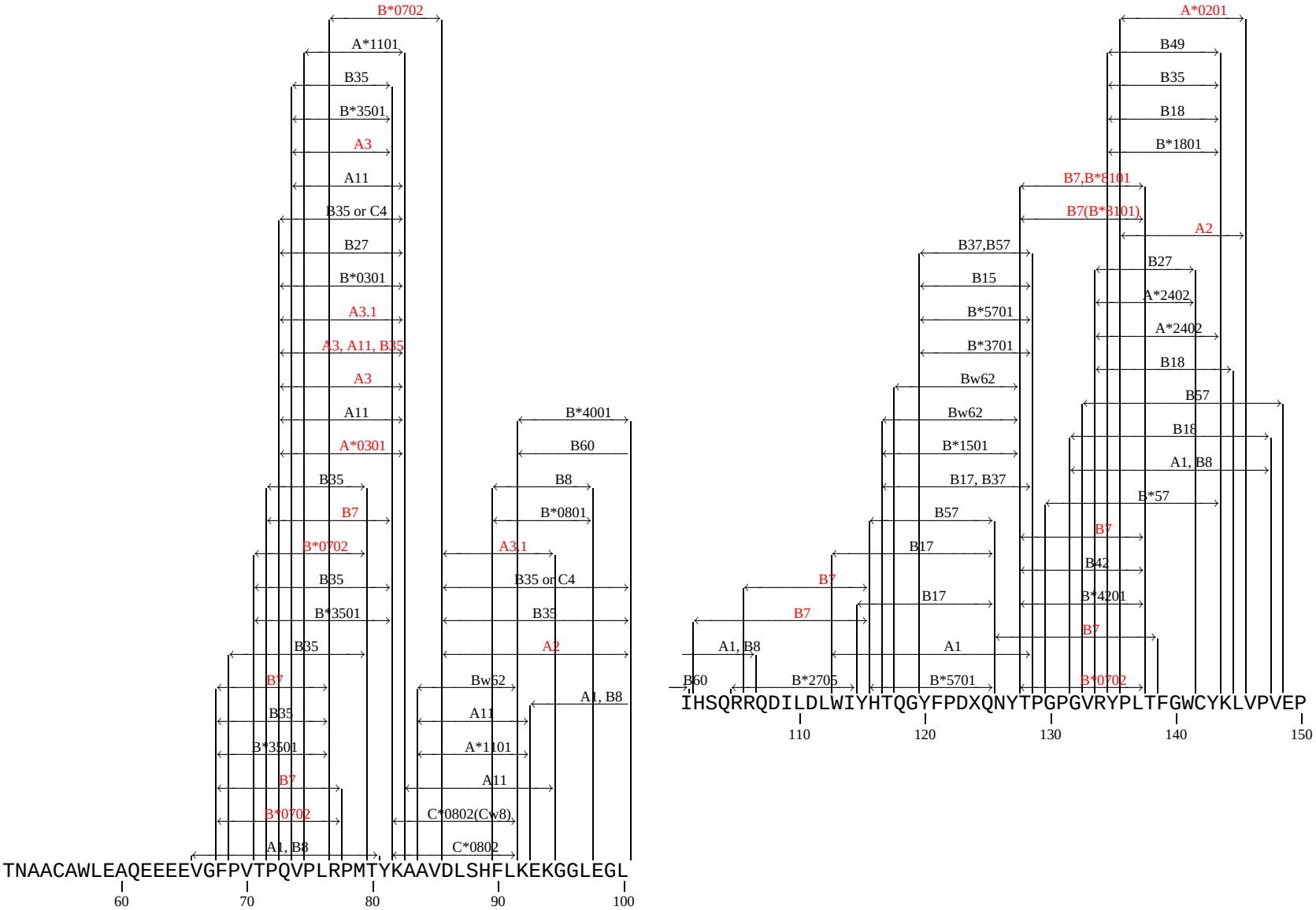


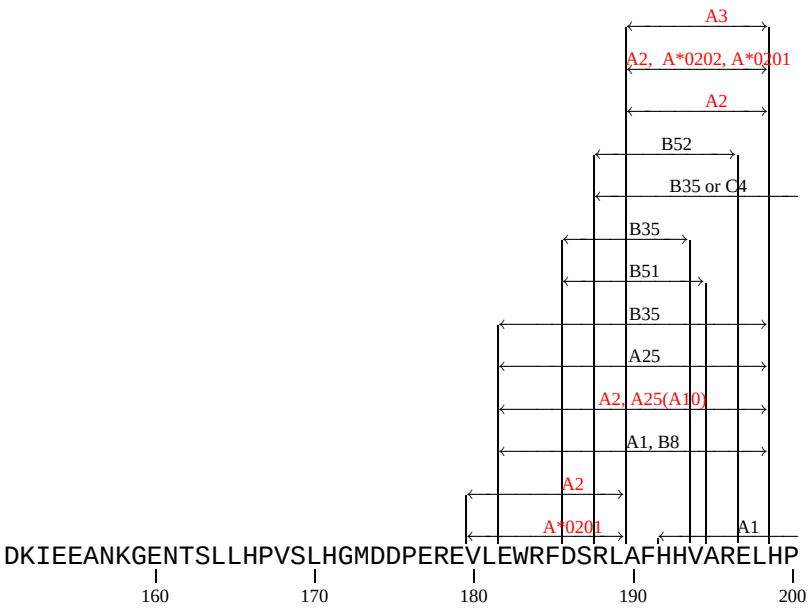
LERILL

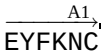
-> gp41 end

Nef CTL Map




B*0702
A*1101
B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
B35
B*3501
B7
B*0702
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A1, B8
A11
A*1101
A11
C*0802(Cw8)
C*0802
TNAACAWLEAQEEEEVGFPVTPQVPLRPMTYKAAVDLSHFLKEKGGLEGL
60
70
80
90
100
A*0201
B49
B35
B18
B*1801
B7,B*8101
B7(B*8101)
A2
B37,B57
B27
B15
A*2402
B*5701
A*2402
B*3701
B18
Bw62
B57
Bw62
B18
B*1501
A1, B8
B17, B37
B*57
B57
B7
B17
B42
B7
B17
B*4201
B7
B7
A1, B8
A1
B60
B*2705
B*5701
B*0702
IHSQRRQDILDLWIYHTQGYFPDXQNYTPGPGVRYPLTFGWCYKLVPVEP
110
120
130
140
150





[Achour (1996)] A. Achour, F. Bex, P. Hermans, A. Burny, & D. Zagury. Induction of anti-gp160 cytotoxic T cells cross-reacting with various V3 loop P18 peptides in human immunode£ciency virus type 1 envelope-immunized individuals. *J Virol* **70**:6741–6750, 1996. (Medline: 96386561).

[Alexander-Miller (1996)] M. A. Alexander-Miller, K. C. Parker, T. Tsukui, C. D. Pendleton, J. E. Coligan, & J. A. Berzofsky. Molecular analysis of presentation by HLA-A2.1 of a promiscuously binding V3 loop peptide from the HIV-1 Envelope protein to human cytotoxic T lymphocytes. *Int Immunol* **8**:641–649, 1996. (Medline: 96324787).

[Bauer (1997)] M. Bauer, M. Lucchiari-Hartz, R. Maier, G. Haas, B. Autran, K. Eichmann, R. Frank, B. Maier, & A. Meyerhans. Structural constraints of HIV-1 Nef may curtail escape from HLA-B7-restricted CTL recognition. *Immunol Lett* **55**:119–22, 1997. (Medline: 97289021).

[Betts (2000)] M. R. Betts, J. P. Casazza, B. A. Patterson, S. Waldrop, W. Trigona, T.-M. Fu, F. Kern, L. J. Picker, & R. A. Koup. Putative immunodominant human imunode£ciency virus-speci£c CD8+ T cell responses cannot be predicted by major histocompatibility complex class I haplotype. *J Virol* **74**:9144–9151, 2000. (Medline: 20438112).

[Birk (1998)] M. Birk, A. Vahlne, A. Sonnerborg, & M. Sallberg. Nonsynonymous mutations within the human immunode£ciency virus type 1 p17 gene are clustered to sequences binding to the host human leukocyte antigen class I molecules. *AIDS Res Hum Retroviruses* **14**:241–8, 1998. (Medline: 98150878).

[Brander (1996)] C. Brander, G. Corradin, T. Hasler, & W. Pichler. Peptide immunization in humans: a combined CD8+/CD4+ T cell-targeted vaccine restimulates the memory CD4 T cell response but fails to induce cytotoxic T lymphocytes (CTL). *Clin Exp Immunol* **105**:18–25, 1996. (Medline: 96280772).

[Brander & Goulder(2001)] C. Brander & P. Goulder. The evolving £eld of HIV CTL epitope mapping: New approaches to the identi£cation of novel epitopes. *HIV Molecular Immunology Database* pages IV–1, 2001. Notes: This review article in the annual HIV Molecular Immunology Compendium presents the table of Optimal CTL Epitopes that has been curated by Brander and others for several years.

[Brander (1995)] C. Brander, W. J. Pichler, & G. Corradin. Identi£cation of HIV-protein derived CTL epitopes for their potential use as synthetic vaccine. *Clin Exp Immunol* **101**:107–113, 1995. (Medline: 95347061).

[Brander & Walker(1995)] C. Brander & B. Walker. The HLA-class I restricted CTL Response in HIV-1 Infection: Identi£cation of optimal epitopes. *HIV Molecular Immunology Database* pages IV–1 to IV–9, 1995.

[Brander & Walker(1996)] C. Brander & B. Walker. The HLA-class I restricted CTL response in HIV-1 Infection: Systematic identi£cation of opti-

mal epitopes. *HIV Molecular Immunology Database* pages IV–50 to IV–60, 1996.

[Brodie (1999)] S. J. Brodie, D. A. Lewinsohn, B. K. Patterson, D. Jiyamapa, J. Krieger, L. Corey, P. D. Greenberg, & S. R. Riddell. In vivo migration and function of transferred HIV-1-speci£c cytotoxic T cells [see comments]. *Nat Med* **5**:34–41, 1999. (Medline: 99098306).

[Brodie (2000)] S. J. Brodie, B. K. Patterson, D. A. Lewinsohn, K. Diem, D. Spach, P. D. Greenberg, S. R. Riddell, & L. Corey. HIV-speci£c cytotoxic T lymphocytes traf£c to lymph nodes and localize at sites of HIV replication and cell death. *J Clin Invest* **105**:1407–17, 2000. (Medline: 20273932).

[Cao (1997)] H. Cao, P. Kanki, J. L. Sankale, A. Dieng-Sarr, G. P. Mazzara, S. Kalams, B. Korber, S. M'Boup, & B. D. Walker. CTL cross-reactivity among different HIV-1 clades: Implications for vaccine development. *J Virol* **71**:8615–8623, 1997. (Medline: 98001384).

[Dadaglio (1991)] G. Dadaglio, A. Leroux, P. Langlade-Demoyen, E. M. Bahraoui, F. Traincard, R. Fisher, & F. Plata. Epitope recognition of conserved HIV envelope sequences by human cytotoxic T lymphocytes. *J Immunol* **147**:2302–2309, 1991. (Medline: 92013025) Notes: Using synthetic peptides, six conserved epitopes on gp120 Env were identi£ed, recognized by polyclonal human CTL in association with HLA-A2 class I. Conserved epitopes: RIQRGPGRAFVTIGK, IIIB; LWVTVYYGVPVWKEATTTLFCA; TTSYTLTSCNTSVITQACPK; SVEINCTRPNNNTRKSI; PEIVTHS; KNCGGEFFYCNS; LPCRIKQFINMWQEVGKAMY; VKIEPLGVAPTKAKRRVVQR. Control: gag, YKRWIILGLNKIVRMYSPT, HLA B27.

[Dorrell (1999)] L. Dorrell, T. Dong, G. S. Ogg, S. Lister, S. McAdam, T. Rostron, C. Conlon, A. J. McMichael, & S. L. Rowland-Jones. Distinct recognition of non-clade B human immunode£ciency virus type 1 epitopes by cytotoxic T lymphocytes generated from donors infected in Africa. *J Virol* **73**:1708–14, 1999. (Medline: 99099071).

[Dupuis (1995)] M. Dupuis, S. K. Kundu, & T. C. Merigan. Characterization of HLA-A*0201-restricted cytotoxic T cell epitopes in conserved regions of the HIV type 1 gp160 protein. *J Immunol* **155**:2232–2239, 1995. (Medline: 95363191) Notes: Five HLA-A2 HIV-1 seropositive HIV-1 MN rec gp160 vaccinees had their CTL activity assessed using peptides known to bind with high af£nity to HLA-A*0201. Four of the patients had speci£c CTL activity for a minimum of at least three epitopes, thus the response appears heterogeneous. One of the four peptides was con£rmed to be HLA A2 restricted. Epitopes were highly conserved.

[Durali (1998)] D. Durali, J. Morvan, F. Letourneur, D. Schmitt, N. Guegan, M. Dalod, S. Saragosti, D. Sicard, J. P. Levy, & E. Gomard. Cross-reactions between the cytotoxic T-lymphocyte responses of human immunode£ciency virus-infected African and European patients. *J Virol* **72**:3547–53, 1998. (Medline: 98216712).

[Ferris (1999)] R. L. Ferris, C. Hall, N. V. Sipsas, J. T. Safrit, A. Trocha, R. A. Koup, R. P. Johnson, & R. F. Siliciano. Processing of HIV-1 envelope glycoprotein for class I-restricted recognition: dependence on TAP1/2 and mechanisms for cytosolic localization. *J Immunol* **162**:1324–32, 1999. (Medline: 99138809).

[Garboczi (1992)] D. N. Garboczi, D. T. Hung, & D. C. Wiley. HLA-A2-peptide complexes: refolding and crystallization of molecules expressed in Escherichia coli and complexed with single antigenic peptides. *Proc Natl Acad Sci USA* **89**:3429–3433, 1992. (Medline: 92228799).

[Goulder (1997a)] P. Goulder, D. Price, M. Nowak, S. Rowland-Jones, R. Phillips, & A. McMichael. Co-evolution of human immunode£ciency virus and cytotoxic T-lymphocyte responses. *Immunol Rev* **159**:17–29, 1997a. (Medline: 98078460).

[Goulder (1997b)] P. Goulder, A. Sewell, D. Lalloo, D. Price, J. Whelan, J. Evans, G. Taylor, G. Luzzi, P. Giangrande, R. Phillips, & A. J. McMichael. Patterns of immunodominance in HIV-1-speci£c cytotoxic T lymphocyte responses in two human histocompatibility leukocyte antigens (HLA)-identical siblings with HLA-A*0201 are in¤uenced by epitope mutation. *J Exp Med* **8**:1423–33, 1997b. (Medline: 97272078) Notes: Primary human immunode£ciency virus (HIV) infection is controlled principally by HIV-speci£c cytotoxic T lymphocytes (CTL) to a steady- state level of virus load, which strongly in¤uences the ultimate rate of progression to disease. Epitope selection by CTL may be an important determinant of the degree of immune control over the virus. This report describes the CTL responses of two HLA-identical hemophiliac brothers who were exposed to identical batches of Factor VIII and became seropositive within 10 wk of one another. Both have HLA-A*0201. The CTL responses of the two siblings were very dissimilar, one donor making strong responses to two epitopes within p17 Gag (HLA-A*0201-restricted SLYNTVATL and HLA-A3-restricted RLRPGGKKK). The sibling responded to neither epitope, but made strong responses to two epitopes presented by HLA-B7. This was not the result of differences in presentation of the epitopes. However, mutations in both immunodominant epitopes of the p17 Gag responder were seen in proviral sequences of the nonresponder. We then documented the CTL responses to two HLA-A*0201-restricted epitopes, in Gag (SLYNTVATL) and Pol (ILKEPVHGV) in 22 other HIV-infected donors with HLA-A*0201. The majority (71%) generated responses to the Gag epitope. In the 29% of donors failing to respond to the Gag epitope in standard assays, there was evidence of low frequency memory CTL responses using peptide stimulation of PBMC, and most of these donors also showed mutations in or around the Gag epitope.

[Goulder (2000a)] P. J. Goulder, C. Brander, K. Annamalai, N. Mngqundaniso, U. Govender, Y. Tang, S. He, K. E. Hartman, C. A. O'Callaghan, G. S. Ogg,

M. A. Altfeld, E. S. Rosenberg, H. Cao, S. A. Kalams, M. Hammond, M. Bunce, S. I. Pelton, S. A. Burchett, K. McIntosh, H. M. Coovadia, & B. D. Walker. Differential narrow focusing of immunodominant human immunode£ciency virus gag-speci£c cytotoxic T-lymphocyte responses in infected African and caucasoid adults and children. *J Virol* **74**:5679–90, 2000a. (Medline: 20283828).

[Goulder (2000b)] P. J. Goulder, Y. Tang, S. I. Pelton, & B. D. Walker. HLA-B57-Restricted cytotoxic T-lymphocyte activity in a single infected subject toward two optimal epitopes, one of which is entirely contained within the other. *J Virol* **74**:5291–9, 2000b. (Medline: 20261752).

[Goulder (1997c)] P. J. R. Goulder, R. E. Phillips, R. A. Colbert, S. McAdam, G. Ogg, M. A. Nowak, P. Giangrande, G. Luzzi, B. Morgan, A. Edwards, A. McMichael, & S. Rowland-Jones. Late escape from an immunodominant cytotoxic T-lymphocyte response associated with progression to AIDS. *Nature Med* **3**:212–216, 1997c. (Medline: 97170968) Notes: The CTL response was studied in six HIV+ individuals who make a strong immunodominat response to the same B27 epitope. In two donors an escape mutation arose after close to 10 years of epitope stability, around the time of progression to AIDS.

[Hadida (1995)] F. Hadida, G. Haas, G. Zimmermann, A. Hosmalin, R. Spohn, A. Samri, G. Jung, P. Debre, & B. Autran. CTLs from lymphoid organs recognize an optimal HLA-A2 restricted and HLA-B52 restricted nonapeptide and several epitopes in the C-terminal region of HIV-1 Nef. *J Immunol* **154**:4174–4186, 1995. (Medline: 95221926) Notes: An *in vitro* limiting dilution analysis showed CTL recognition in the context of HLA B52 and A2.1, A2.2 and A2.4 in nanomolar concentrations. Molecular modeling suggests motifs important for peptide binding to the pocket of an HLA-A2.1 molecule.

[Hammond (1995)] S. A. Hammond, R. P. Johnson, S. A. Kalams, B. D. Walker, M. Takiguchi, J. T. Safrit, R. A. Koup, & R. F. Siliciano. An epitope-selective transporter associated with antigen presentation TAP-1/2-independent pathway and a more general TAP-1/2-dependent antigen-processing pathway allow recognition of the HIV-1 envelope glycoprotein by CD8+ CTL. *J Immunol* **154**:6140–6156, 1995. (Medline: 95271010) Notes: Two peptide-processing pathways are utilized for MHC class I presentation of HIV-1 Env epitopes. The previously characterized TAP-1 and TAP-2 dependent pathway can generate all Env epitopes and uses Env protein mislocalized in the cytosol to produce peptides. The second, novel pathway uses a TAP-1/2 independent pathway, and allows a subset of MHC-restricted epitopes to be processed in the endoplasmic reticulum or a Golgi compartment.

[Hickling (1990)] J. K. Hickling, C. M. Fenton, K. Howl and , S. G. Marsh, & J. B. Rothbard. Peptides recognized by class I restricted T cells also bind to MHC class II molecules. *International Immunology* **2**:435–441, 1990. (Medline: 91197875) Notes: Peptides shown to be presented in the context of MHC class I proteins by mouse or human CD8+ T lymphocytes could also bind to HLA-DR molecules on the surface of B lymphoblastoid cell lines (B-LCL). Four out of £ve class I-restricted T cell determinants bound, including the HIV-1 gp120 epitope.

[Jin (2000)] X. Jin, C. G. Roberts, D. F. Nixon, J. T. Safrit, L. Q. Zhang, Y. X. Huang, N. Bhardwaj, B. Jesdale, A. S. DeGroot, & R. A. Koup. Identi£cation of subdominant cytotoxic T lymphocyte epitopes encoded by autologous HIV type 1 sequences, using dendritic cell stimulation and computer-driven algorithm. *AIDS Res Hum Retroviruses* **16**:67–76, 2000. (Medline: 20092440).

[Kaul (2000)] R. Kaul, F. A. Plummer, J. Kimani, T. Dong, P. Kiama, T. Rostron, E. Njagi, K. S. MacDonald, J. J. Bwayo, A. J. McMichael, & S. L. Rowland-Jones. HIV-1-speci£c mucosal CD8+ lymphocyte responses in the cervix of HIV-1- resistant prostitutes in Nairobi. *J Immunol* **164**:1602–11, 2000. (Medline: 20109119).

[Kmieciak (1998)] D. Kmieciak, I. Bednarek, M. Takiguchi, T. J. Wasik, J. Bratosiewicz, A. Wierzbicki, H. Teppler, J. Pientka, S. H. Hsu, Y. Kaneko, & D. Kozbor. The effect of epitope variation on the pro£le of cytotoxic T lymphocyte responses to the HIV envelope glycoprotein. *Int Immunol* **10**:1789–99, 1998. (Medline: 99100990).

[Kundu (1998a)] S. K. Kundu, M. Dupuis, A. Sette, E. Celis, F. Dorner, M. Eibl, & T. C. Merigan. Role of preimmunization virus sequences in cellular immunity in HIV- infected patients during HIV type 1 MN recombinant gp160 immunization. *AIDS Res Hum Retroviruses* **14**:1669–78, 1998a. (Medline: 99085868).

[Kundu (1998b)] S. K. Kundu, E. Engleman, C. Benike, M. H. Shapero, M. Dupuis, W. C. van Schooten, M. Eibl, & T. C. Merigan. A pilot clinical trial of HIV antigen-pulsed allogeneic and autologous dendritic cell therapy in HIV-infected patients. *AIDS Res Hum Retroviruses* **14**:551–60, 1998b. (Medline: 98252383).

[McKinney (1999)] D. McKinney, D. Lewinson, S. Riddell, P. Greenberg, & D. Mosier. The antiviral activity of HIV-speci£c CD8+ CTL clones is limited by elimination due to encounter with HIV-infected targets. *J. Immuno* **163**:861–7, 1999. (Medline: 99323981).

[McMichael & Walker(1994)] A. J. McMichael & B. D. Walker. Cytotoxic T lymphocytes epitopes: implications for HIV vaccine. *AIDS* **8S**:S155–S173, 1994. Notes: Comprehensive review summarizing CTL epitopes that have known HLA type and are £ne mapped to indicate epitope boundaries. Anchor residues are indicated when known for different HLA restricted epitopes. Includes a summary of the published literature, as well as much work that was in press or submitted for publication.

[Menendez-Arias (1998)] L. Menendez-Arias, A. Mas, & E. Domingo. Cytotoxic T-lymphocyte responses to HIV-1 reverse transcriptase (review). *Viral Immunol* **11**:167–81, 1998. (Medline: 99203068).

[Parker (1992)] K. C. Parker, M. A. Bednarek, L. K. Hull, U. Utz, B. C. H. J. Zweerink, W. E. Biddison, & J. E. Coligan. Sequence motifs important for peptide binding to the human MHC class I molecule, HLA-A2. *J Immunol* **149**, 1992. (Medline: 93056532).

[Rowland-Jones (1998a)] S. Rowland-Jones, T. Dong, P. Krausa, J. Sutton, H. Newell, K. Ariyoshi, F. Gotch, S. Sabally, T. Corrah, J. Kimani, K. MacDonald, F. Plummer, J. Ndinya-Achola, H. Whittle, & A. McMichael. The role of cytotoxic T cells in HIV infection. *Dev Biol Stand* **92**:209–14, 1998a. (Medline: 98214896) Notes: In this paper CTL response to previously de£ned conserved epitopes was found in exposed but uninfected prostitutes in Nairobi. Subtypes A and D are circulating in this regions, and the reactive epitopes tended to be conserved. Similarly previous studies in the Gambia showed that exposed but uninfected prostitutes tended to have B35 presented CTL epitopes conserved between HIV-1 and HIV-2. It was suggested that what was special about B35 is simply that it presents epitopes found both in HIV-1 and HIV-2.

[Rowland-Jones (1998b)] S. L. Rowland-Jones, T. Dong, K. R. Fowke, J. Kimani, P. Krausa, H. Newell, T. Blanchard, K. Ariyoshi, J. Oyugi, E. Ngugi, J. Bwayo, K. S. MacDonald, A. J. McMichael, & F. A. Plummer. Cytotoxic T cell responses to multiple conserved HIV epitopes in HIV- resistant prostitutes in Nairobi [see comments]. *J Clin Invest* **102**:1758–65, 1998b. (Medline: 99021675).

[Safrit (1994)] J. T. Safrit, A. Y. Lee, C. A. Andrews, & R. A. Koup. A region of the third variable loop of HIV-1 gp120 is recognized by HLA-B7-restricted CTLs from two acute seroconversion patients. *J Immunol* **153**:3822–3830, 1994. (Medline: 95015873) Notes: HIV-1 envelope-speci£c CTL clones were isolated from the peripheral blood of two patients within weeks of seroconversion. These clones were CD8+ and restricted by the HLA-B7 molecule. The minimum epitope was de£ned, RPNNNTRKSI, with anchor residues at the proline and isoleucine; the anchor residues are relatively well conserved. A Serine to Arginine change at position 9 of the epitope abrogated clone recognition in one of the patients. This amino acid change is one factor that has been associated with a change from a nonsyncytium-inducing to a syncytium-inducing phenotype of HIV-1.

[Takahashi (1991)] K. Takahashi, L.-C. Dai, T. R. Fuerst, W. E. Biddison, P. L. Earl, B. Moss, & F. A. Ennis. Speci£c lysis of human immunode£ciency virus type 1-infected cells by a HLA-A3.1-restricted CD8+ cytotoxic T-lymphocyte clone that recognizes a conserved peptide sequence within the gp41 subunit of the envelope protein. *Proc Natl Acad Sci USA* **88**:10277–10281, 1991. (Medline: 92052253) Notes: gp41 epitope: RLRDLLLIVTR, HLA A3.1 (NL43). Synthetic peptides of RF and CDC4 were recognized by CTL clone despite non-conservative Thr to (Val or Ala) change, but an MN peptide with four natural substitutions was not recognized.

[van der Burg (1997)] S. H. van der Burg, M. R. Klein, O. Pontesilli, A. M. Holwerda, J. Drijfhout, W. M. Kast, F. Miedema, & C. J. M. Melief. HIV-1 reverse transcriptase-speci£c CTL against conserved epitopes do not protect against progression to AIDS. *J Immunol* **159**:3648–3654, 1997. (Medline: 97461484).

[Wilkens & Ruhl(1999)] B. Wilkens & D. Ruhl. Personal communication 1999.

[Wilson (1996)] C. Wilson, B. Wilkes, D. Ruhl, & B. Walker. Personal communication. 1996. Notes: De£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study. Personal communication.

[Wilson (1999)] C. C. Wilson, R. C. Brown, B. T. Korber, B. M. Wilkes, D. J. Ruhl, D. Sakamoto, K. Kunstman, K. Luzuriaga, I. C. Hanson, S. M. Widmayer, A. Wiznia, S. Clapp, A. J. Ammann, R. A. Koup, S. M. Wolinsky, & B. D. Walker. Frequent detection of escape from cytotoxic T-lymphocyte recognition in perinatal human immunode£ciency virus (HIV) type 1 transmission: the ariel project for the prevention of transmission of HIV from mother to infant. *J Virol* **73**:3975–85, 1999. (Medline: 99214336).

[Wilson (2000)] J. D. Wilson, G. S. Ogg, R. L. Allen, C. Davis, S. Shaunak, J. Downie, W. Dyer, C. Workman, S. Sullivan, A. J. McMichael, & S. L. Rowland-Jones. Direct visualization of HIV-1-speci£c cytotoxic T lymphocytes during primary infection. *AIDS* **14**:225–33, 2000. (Medline: 20179241).

[Wilson (1998)] S. E. Wilson, S. L. Pedersen, J. C. Kunich, V. L. Wilkins, D. L. Mann, G. P. Mazzara, J. Tartaglia, C. L. Celum, & H. W. Sheppard. Cross-clade envelope glycoprotein 160-speci£c CD8+ cytotoxic T lymphocyte responses in early HIV type 1 clade B infection. *AIDS Res Hum Retroviruses* **14**:925–37, 1998. (Medline: 98349428).

[Zarling (1999)] A. L. Zarling, J. G. Johnson, R. W. Hoffman, & D. R. Lee. Induction of primary human CD8+ T lymphocyte responses In vitro using dendritic cells. *J Immunol* **162**:5197–204, 1999. (Medline: 99244883).